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THE MANAGEMENT OF ACUTE HEAD INJURIES*

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In a symposium on trauma the discussor of acute head injuries can comment only briefly on each of the more important aspects of the problem. For convenience, the general measures applicable to all cases will be outlined first, and this will be followed by a discussion of the specific treatment of the common types of head injury.

GENERAL MEASURES APPLICABLE TO ALL CASES

1. *First Aid:* Practically speaking, there are no first aid measures applicable to patients with head injury other than the application of an elastic pressure bandage to scalp wounds before transportation of the patient to the nearest hospital or physician's office. However, many patients require first aid treatment for associated injuries, such as fracture of the cervical spine or long bones. Such patients should not be given morphine unless they are in extreme pain.

2. *Treatment of Shock:* Traumatic shock is rare in patients whose injury is limited to the head. If it occurs, it should, of course, be combatted by the administration of blood or plasma. Unless the state of shock is alarming, the injured head should not be lowered below the level of the heart. There is rarely need for immediate suture of the scalp or debridement of compound fractures. Surgical treatment can be deferred twelve or more hours for stabilization of the patient's condition unless there are signs of deterioration due to intracranial bleeding.

3. *History Taking:* Just before or while examining the patient the physician should inquire as to the exact hour of the accident. It is important to find out if the patient became unconscious at the moment of the impact or whether unconsciousness developed minutes or hours later. Often, a fair estimate can be made of the degree of cerebral damage if all details of the accident are known. An interval of consciousness between the primary and secondary attacks of coma usually indicates active bleeding from the middle meningeal artery.

4. *Examination:* Because the unconscious patient with cerebral trauma may also have other serious injuries, such as a ruptured spleen requiring emergency surgical intervention, the preliminary examination should be primarily concerned with a brief search for other injuries. Following this, painful stimuli should be applied to unconscious patients to demonstrate the presence of weakness or paralysis of the facial muscles and extremities, as well as to estimate depth of the coma. The temperature, pulse rate, blood pressure, and character and rate of respiration should be determined. The state of the pupils is important. Ears and nose should be inspected for blood and cere-

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brospinal fluid. Special attention should be given to palpation of the skull and investigation of scalp wounds.

5. *Continuous Observation of Patient:* Since the exact diagnosis in a head injury is never known within the first few days, continuous observation of the patient is absolutely essential. Special nursing care and frequent examinations by the physician are imperative. Change in the character of respiration, rise in blood pressure, increase in temperature above 101° F., fall in pulse rate below 60 beats per minute, development of hemiparesis, deepening of unconsciousness, and dilatation of a pupil must not go uncharted. The patient who becomes less comatose with the passage of time probably does not have a surgical lesion and most likely will survive. Conversely, the patient whose coma becomes progressively deeper presumably has an intracranial hematoma requiring surgical treatment.

6. *Making of Roentgenograms:* It is customary to make stereoscopic roentgenograms of the skull, and often of the neck, shortly after the patient's arrival in the hospital. However, these should not be taken until the physical examination has been done and treatment for shock, if present, has been completed. Patients in poor condition should not be taken to the roentgenologic department until their condition has improved or a decision has been made to operate.

7. *Spinal Puncture:* It is generally considered preferable to omit spinal puncture at the time of the initial examination in patients with acute head injury. Study of the spinal fluid and measurement of the cerebrospinal fluid pressure during the first few hours almost never provides information of therapeutic value. Furthermore, removal of fluid in any patient who has an intracranial hematoma is obviously dangerous. Spinal puncture on the second or third day is sometimes performed for the relief of headache or for the discovery of specific information. For example, is stiffness of the neck due to vertebral injury, subarachnoid hemorrhage, or meningitis? Also, spinal drainage every twelve hours is indicated for cerebrospinal rhinorrhea. It is now generally known that the spinal fluid pressure is normal or subnormal in most patients with cerebral injury in the absence of gross hemorrhage.

8. *Maintenance of Fluid Balance:* Treatment of the unconscious patient with severe brain injury by hypertonic solutions intravenously, magnesium sulphate orally or rectally, and strict limitation of fluid intake has become obsolete. The intravenous administration of concentrated serum albumin, concentrated plasma, or 50 per cent solution of dextrose should be reserved for patients with definite evidence of high intracranial pressure due to hemorrhage or anoxia. In recent years it has been well established that head injury causes little cerebral edema and little if any increase in intracranial pressure. The chief causes of cerebral swelling are anoxia of the blood and intracranial hemorrhage. It is advisable to give an adult from 1500 cc. to 1800 cc. of fluid daily. In the beginning 800 cc. of a 5 per cent solution of dextrose in distilled water and 1000 cc. of sodium chloride in a 5 per cent solution of glucose can be given intravenously. Patients unconscious for more than twenty-four hours should receive food and fluid by gastric tube. If patients are receiving sulfonamides, the daily fluid intake should be at least 2000 cc., regardless of the state of consciousness. A more physiologic approach is to maintain the urinary output at 1000 cc. every twenty-four hours regardless of the fluid intake required.

9. *Management of Urinary Incontinence or Retention:* Unconscious patients who do not void or who are incontinent require indwelling catheterization.

10. *Administration of Sedatives:* Restless, noisy, struggling patients need sedation for at least part of each twenty-four hours but it is wise to withhold such medication until a diagnosis has been made and definitive treatment started. Morphine is to be avoided in patients with head injury except under special circumstances. Sodium phenobarbital or sodium amytal, either

intramuscularly or intravenously, can be given until the desired effect has been obtained. Codeine satisfactorily relieves pain in conscious patients.

11. *Control of Convulsions:* Convulsions in patients with acute head injuries fortunately are uncommon. If several occur within a short time, an adult patient should receive 3 to 5 gr. of sodium phenobarbital intramuscularly. If this does not control the seizures, repeated doses of 3 gr. of sodium phenobarbital should be given intramuscularly or intravenously until the seizures have ceased. Dilantin sodium, 6 gr., should be given by mouth simultaneously and each day thereafter for several days or weeks. One must be fully aware that convulsions, particularly Jacksonian convulsions, may be an early sign of an extradural, subdural, or subcortical clot.

12. *Treatment of Respiratory Difficulties:* Fall of the respiratory rate below 14 per minute, rise above 35, periods of apnea, labored respiration and "moist" respiration are grave prognostic signs in patients with cerebral injuries. If a patient's color or breathing is not perfect, he should receive oxygen by nasal tube at the rate of 6 liters a minute. If partial obstruction of the pharynx or trachea by the tongue or by secretions cannot quickly be remedied by a rubber airway, endotracheal suction, or postural drainage, tracheotomy should be performed. Probably 20 per cent of patients with severe cerebral injuries should have a tracheotomy. The response of these patients to tracheotomy is invariably spectacular. It seems safe to state that maintenance of a wide open and dry tracheobronchial tree is the most important single factor in the treatment of severe injuries of the brain. Cerebral edema is caused by anoxia of the blood, not by cerebral damage.

13. *Use of Antibiotics and Sulfonamides*: In compounds fractures of the skull, of any type, the patient should receive sulfadiazine by mouth, and penicillin intramuscularly, for ten days. Doses of 10,000 units of penicillin can be safely applied locally in solution after debridement of wounds of the scalp or brain, but the local use of sulfonamides is to be avoided.

14. *Control of Fever*: Rising temperature during the first twelve hours in patients with acute cranial injuries indicates damage to the brain stem or irritation from blood in the ventricles and subarachnoid space and is a bad prognostic sign. Some patients die of hyperthermia. Treatment consists of applying enough ice bags, alcohol rubs, and electric fans to keep the rectal temperature at or below 101° F.

15. *Avoidance of Bed Rest*: Patients with any type of head injury, whether surgical or nonsurgical, should be encouraged to sit in a chair and walk as soon as they seem physically able to do so. Maximum reassurance and even apparent indifference to symptoms on the part of the physician in charge is indicated during the convalescent period if post-traumatic psychoneurosis is to be kept in its mildest form.

SPECIFIC TREATMENT OF THE COMMON TYPES OF HEAD INJURY

1. *Cerebral Concussion*: For practical purposes, a diagnosis of simple cerebral concussion is made if the patient regains consciousness within fifteen minutes. Longer periods of unconsciousness indicate that there has also been contusion of the brain. No special treatment is indicated for concussion but close observation of the patient for eighteen hours is essential because of the remote possibility of bleeding from a ruptured middle meningeal artery. It might be pointed out here that the long-standing theory that there are no microscopic changes in the brain after concussion has been abandoned. Windle, Groat and Fox[1] have shown that in animals with concussion certain microscopic changes in brain cells can be demonstrated after a lapse of two to eight days. This structural basis for the symptoms of concussion has been accepted by Denny-Brown[2] and other authorities.

2. *Simple Linear Fracture of Skull*: Linear fractures of the skull can occur with or without loss of consciousness. The diagnosis is made by roentgenography. Gener-

ally speaking, no treatment is required unless the fracture causes a tear of the middle meningeal artery. A linear fracture which extends into a paranasal sinus is actually a compound fracture of the skull and should be treated accordingly.

3. *Simple Depressed Fractures of Skull*: Most simple, depressed fractures of the skull are incapable of exerting continued pressure on the underlying brain and require no treatment. Patients with severely depressed fractures should be transferred to a neurosurgeon several days folowing the accident after the immediate effects of the injury have subsided. Excision of the depressed fragments and repair of the defect with a tantalum plate is the treatment of choice, although occasionally the depressed fragments can be restored to their normal position.

4. *Lacerations of the Scalp*: Lacerations of the scalp should be sutured immediately or, if more convenient, six to twelve hours later. Shaving the scalp about the laceration and thoroughly cleansing the wound with soap and water are essential. Sulfonamides should not be placed in such wounds. Closure without drainage is accomplished with interrupted sutures of cotton or silk. Subcutaneous sutures are to be avoided in most instances. The application of a pressure bandage in extensive lacerations will prevent the formation of a hematoma under the scalp.

5. *Compound Linear Fractures of the Skull*: If roentgenograms show that a linear fracture extends into a paranasal sinus, or if a linear fracture of the skull is noted during repair of a laceration of the scalp, the patient should receive an intramuscular injection of 30,000 units of penicillin. Additional penicillin intramuscularly, and sulfadiazine orally, should be administered for ten days as prophylaxis against infection. Debridement of the fracture line is not indicated.

6. *Extradural Hematoma*: Every patient who sustains a major or minor injury of the head should be suspected of having an extradural hematoma until the passage of time convinces the observer of the contrary. This means that the patient must be watched closely for eighteen hours. The patient should not be left alone. The pulse and respiratory rates should be checked every fifteen minutes. A sleeping patient should be aroused at least every hour to make certain that he has not slipped into coma. When bradycardia, slow respiration, vomiting, deepening coma, inequality of pupils, Jacksonian seizures, or hemiparesis call attention to bleeding from a torn middle meningeal artery, a large opening must be made in the temporal bone as quickly as possible. There is not time to send the patient to a neurosurgeon. Blood transfusions are needed.

7. *Compound Depressed Fractures of the Skull and Penerating Wounds of the Brain*: Patients with these types of injuries should receive penicillin and streptomycin and be transferred to a hospital where a neurosurgeon is available. These patients tolerate ambulance and air travel. Twenty-four hours is early enough for surgical treatment in most cases. The anesthetic of choice is procaine infiltration with or without sodium pentothal intravenously. If these are not suitable, the endotracheal administration of ether is indicated. A perfect airway is essential at all times and one should not hesitate to employ endotracheal intubation or to perform tracheotomy. The treatment consists of debridement of the scalp, skull and brain, absolute hemostasis, perhaps the local application of a solution of 10,000 units of penicillin, and closure of the dura. If part of the dura has been destroyed, the defect is repaired with periosteum, temporal fascia, or synthetic membrane. The importance of dural closure and scalp closure without tension cannot be overestimated. Attention should be called to the occasional great usefulness of gelfoam and thrombin in the control of bleeding from cortical vessels.

The traumatic surgeon must be aware of the possibility of an intracerebral hematoma or a subdural hematoma at the point of exit of a missile. It is well to excise the wound of exit in the scalp and debride the skull and meninges in most cases.

8. *Cerebrospinal Rhinorrhea*: The escape of spinal fluid from the ear in a basal fracture of the skull almost invariably stops spontaneously within a day or two but cerebrospinal rhinorrhea is a more serious problem. Antibiotics, sulfadiazine, and spinal drainage every twelve hours, usually solve the problem within a few days. If the fistula persists for a week, in spite of spinal drainage, the tear in the dura should be repaired through a frontal osteoplastic craniotomy. Roentgenograms should be repeated in cases of cerebrospinal rhinorrhea to determine whether or not air has entered the subarachnoid space and ventricles.

9. *Severe Brain Injury Without Compound Fracture of Skull*: The majority of serious head injuries fall within this category. Surgical treatment is not indicated unless an extradural, subdural, or intracerebral hematoma is suspected and demontrated by trephination, arteriography, or ventriculography. Patients may make a remarkably satisfactory recovery after several weeks of unconsciousness but during this period there must be special nursing care, intranasal administration of oxygen, adequate food and fluids by gastric tube, indwelling catheterization, measurement of urinary output, aspiration of tracheobronchial tree, often tracheotomy, administration of sulfonamides and antibiotics, frequent change of position, control of fever, and prevention of extreme lestlessness or convulsions.

10. *Acute Subdural Hematoma*: This is not a clinical entity. Often a collection of arterial or venous blood is found in the subdural or subarachnoid space or both during surgical operations for compound fractures of the skull. The associated laceration or penetrating wound of the brain is usually of greater importance than the hematoma.

11. *Subdural Hygroma*: In this relatively rare lesion, a small tear in the arachnoid permits cerebrospinal fluid to enter the potential subdural space where it becomes trapped. Since there is no absorptive mechanism in the subdural space, the patient has signs and symptoms of increasing intracranial pressure. If the collection of subdural fluid becomes sufficiently large, all the signs and symptoms of subdural hematoma may appear. Prompt recovery follows trephination and evacuation of the fluid through an opening in the dura.

12. *Intracerebral Hematoma*: Occasionally trauma, as well as apoplexy, causes a hematoma to form within the substance of the brain. This gives rise, rather rapidly, to the same signs and symptoms that a tumor in the same location would be expected to produce. After localization of such a clot by clinical methods, arteriography or pneumoencephalography, it can usually be removed through a 3 cm. trephine.

13. *Chronic Subdural Hematoma*: Chronic subdural hematoma is a late complication of a blow to the head and does not usually manifest itself until weeks or months after injury. The treatment is complete or partial removal of the lesion through an osteoplastic flap or through one or more trephines.

REFERENCES

1. Windle, W. F., Groat, R. A. and Fox, C. A.: Experimental structural alterations in brain during and after concussion. Surg., Gynec. & Obst. 79:561 (Dec.) 1944.

2. Denny-Brown, D.: Cerebral concussion. Physiol. Rev. 25:296 (April) 1945.

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EARLY MANAGEMENT OF INJURIES OF THE CHEST*

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SHREVEPORT

The purpose of this paper is to discuss the newer as well as the older first aid measures in lifesaving in injuries of the chest. Injuries of the chest are often very serious emergencies. The heart and lungs receive injuries with less physiological quietude than almost any other part of the body. An analysis of 11,000,000 English, French, American, and German wounded

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revealed only 6 per cent had chest wounds, but of the total mortality of 8 per hundred, 56 per cent died from chest wounds.[1] The patient with a chest wound has the same shock, hemorrhage, and sepsis as those with wounds elsewhere in the body, and, in addition, he may have interference with heart and lung action. He may have compression of the lung, or lungs, with blood or air in the chest cavity. He may have a sucking wound or open pneumothorax. He may have pressure on the heart from excessive blood or air in the chest, or blood in the pericardial sac itself. Early relief of these physiological grievances is often lifesaving. It is not the work of the specialist but of the quick thinking, informed family doctor at the time of accident that saves the greatest number of lives.

Close inspection and timely observations of the patient are needed before planning the treatment. Wrong treatment is often worse than no treatment at all. Are the patient's finger nails blue? Is he cold and clammy, or is he warm and moist? Is his pulse full and bounding, or is it thin and thready? When did he get hurt? Do both sides of his chest move equally? Where is the heart beat? Is he spitting or coughing up blood? Does he wheeze? Is his wound sucking air? All of these simple things are the guide posts to treatment.

The "firsts" in treatment are directed towards saving life. If he is blue give him oxygen—yes, from the welder's oxygen tank if no other is immediately available. If he is in shock give him fluids as well as oxygen. Any sterile carbohydrate or plasma solution is good until whole blood can be obtained. Put his head lower than his body and keep him warm. These were and still are good procedures to save life. Once these first aid measures are done check his chest for movement. If one side of his chest does not move, then he has either fractured ribs, or air or blood in his chest. Percuss it. If flat, it is blood, and if resonant, it is air. If resonant, he may have a pneumothorax or he may have fractured ribs limiting the chest action. Listen for breath sounds. If they are absent it is a pneumothorax. Note the location of the heart beat. If it is out of place or he has a weak pulse pressure, and the neck veins bulge, he has serious impairment to the return blood supply to the heart. If his pressure is low he has either a shift of the mediastinum or fluid in the pericardial sac. A decision between the two is made from the nature of the injury and the pulse pressure. If there is a mediastinal shift, take off air or fluid by thoracentesis. If there is continued leaking of air and evidence of mediastinal shift recurs, insert a blunted large needle or install an intercostal rubber catheter in the fifth rib interspace in the midaxillary line and attach it to a water bottle trap on the floor. This will promote gradual re-expansion of the lung. If blood is found and the injury is only a few hours old, replace half of the blood removed with air. If a large pulmonary vessel is injured he may start bleeding again. Autotransfusion may be used when it is expedient. Injuries to the intercostal vessels in penetrating wounds of the chest are much less common than injuries to the pulmonary vessels themselves. The lung tends to control intrapulmonary bleeding itself, if not re-expanded too vigorously and too early. If pericardial fluid is found a pericardial aspiration may need to be done.

If the patient has obstruction in the throat or trachea get a clear airway. A blow with the palm of the hand on the sides of the chest will often clear a larynx of obstruction. An ordinary uretheral catheter attached to the office nasal suction apparatus can be used harmlessly, and often as effectively as the bronchoscope. Pull the patient's tongue forward with a cotton sponge and insert the catheter through the nose so that the tip is in the posterior pharynx. When he inhales plunge the catheter. It may not go into his trachea the first try, but it will the second or third attempt. Suck the trachea dry. The catheter will not only clear the trachea but will stimulate coughing and thus help to clear his bronchial tubes as well.

Fractured ribs are probably the most common chest injury. A simple fracture

of one is of only painful moment. A compound fracture may be lethal. You do not need a modern x-ray apparatus, just listen to the chest with an old fashioned stethoscope. If no breath sounds are heard it is more serious. Unless the patient is a very fat person check his ribs with your "doctor's" fingers. If he is conscious, press the ribs near the vertebral column and have him indicate the point of pain. The fracture is there where he indicates. Many older text books and doctors advocate strapping the chest with adhesive tape in rib fractures. This has some danger. It is better not to do it. Lacerations of a sound lung may result just from the pressure, and enough interference with breathing may be produced to promote atelectasis and its sequel pneumonia. Who has not observed pneumonia in rib fracture cases. A safer and more pain allaying method is to inject the tissues around the fracture site with novocain. One injection may well stop the spasm and pain for many hours. Re-inject if necessary.[2]

If there is a compound fracture from a penetrating wound and there is sucking of air, treat the sucking wound by a compression dressing.[3] Later treat the fracture. It may require the training and skill of the chest surgeon, particularly if there is marked injury to the intercostal vessels or lung. Multiple fractures of ribs some times require considerable surgery and much care postoperatively. If subcutaneous or body cavity emphysema is present give oxygen at 10 to 14 liters per minute. Use a nasal catheter or a Boothby-Sandiford mask. Stop the oxygen in two hours. Then two hours later start it again. Marked relief will be noticed. One or two such treatments are often sufficient. This is a lifesaving measure in severe mediastinal emphysema.

Chemical injuries may be grouped in three classes: dust particles, gases and oils. Pneumoconiosis is not a surgical problem. Gases may be. In mild cases fresh air is good treatment. Nasal or mask oxygen may be used in the more severe cases. If the patient is comatose then artificial respiration must be used with the above. Oils may require specific treatment. In rural areas the accidental aspiration of kerosene is common and tractor fuel tanks frequently contribute to the tractor operators getting fuel into their tracheas. Early treatment is supportive. If any large amount is present then postural drainage and aspiration with a catheter are indicated. Aminophyllin may help dilate edematously obstructed bronchi. Bronchoscopy with direct application of adrenalin to edematous bronchial mucosa may be necessary. Chemotherapy should be instituted to prevent pneumonia.

Missiles that penetrate the chest and abdominal cavities simultaneously may injure the liver. When shock exists, treat it by the usual supportive methods. When shock has been controlled the patient should be moved to a surgical hospital. If he has both a chest bleeding wound and an abdominal bleeding wound then all available facilities will be needed at operation. The blood bank and the specialists will then both need to be on hand.

Injuries to the brachial plexus and brachial blood vessels are occasionally observed in gunshot wounds of the chest. Any palsy of the arms or hands is significant. Call it to the attention of the patient and bystanders before surgical treatment. Brachial blood vessels and subclavian vessels when injured often develop aneurysms. These as well as nerve injuries may be treated surgically at a later date. Some of them do not require any treatment at all other than "time."

Artificial respiration should be given with deliberation and caution. Many respirators produce too high a pressure for the lungs. Actual rupture of the lung parenchyma has been produced by such instruments. Mediastinal emphysema has been produced resulting in marked pressure on the vena cava and death.[4] No pressure over 20 cm. of water should ever be used.[5] In the presence of fractured ribs manual artificial respiration should be done with great caution or not at all. Actual puncture of the lung has been done by manual artificial respiration in the presence of frac-

tured ribs. Oxygen mask apparatus is to be preferred whenever available.

Important secondary measures after immediate lifesaving procedures have been instituted are chemotherapeutic administrations. Either 300,000 units of penicillin or 1 gram of streptomycin or both should be given parenterally within a few hours after the injury. Infection in chest wounds may be extremely serious. If the skin is broken antitetanic serum should be given. External dressings of furacin soluble dressing have proved extremely effective in combating infection in external abrasions and lacerations.

CONCLUSIONS

1. Injuries of the chest generally require earlier well directed systematic first aid treatment than wounds elsewhere in the body.

2. Consideration for cardiac and respiratory function must be foremost in planning first aid treatment.

3. Careful inspection of the chest action is important in planning early management.

4. Treat the shock first.

5. Artificial respiration should be given with great caution.

6. Chemotherapeutic agents should be given all chest injuries early.

7. Definitive surgical treatment is best done in a hospital where there is a blood bank and a staff of experienced surgeons conversant with the chest.

REFERENCES

1. Berry, Frank B.: The treatment of injuries to the chest. Am. J. Surg., 54:280, 1941.

2. Harmon, Paul H., Baker, Dan R. and Kornegay, Robert D.: Uncomplicated fractures of ribs and major injuries of the chest wall. Treatment by infiltration with local anesthetic. J. A. M. A., 118:30, 1942.

3. Shefts, Lawrence M. and Doud, Ernest A.: The management of thoracic and thoraco-abdominal wounds in the forward areas in the Sicilian and Italian campaigns. J. Thoracic Surg., 15:205, 1941.

4. Adams, W. E.: Differential pressures and reduced lung function in intra-thoracic operations. J. Thoracic Surg., 9:254, 1940.

5. Marcotte, R. J., Phillips, F. J., Adams, W. E. and Livingstone, H.: Differential intrabronchial pressures and mediastinal emphysema. J. Thoracic Surg., 9:346, 1940.

NONPENETRATING INJURIES OF THE ABDOMEN

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Serious injuries of abdominal viscera resulting from forces that do not penetrate or perforate the abdominal wall are not common. Yet it seems proper to consider these injuries at this time, because the majority of them are acquired in vehicular accidents. We may expect more accidents during the summer months than during the winter months. Because of infrequency of occurrence, lack of identifying early symptoms, apparent rapid recovery from severe trauma, disastrous effects from what seems to be mild injury, the prognosis is grave and the mortality exceedingly high. In at least 25 per cent of our series and that of Rowlands[1] there were other injuries that made the abdominal injuries more difficult to diagnose and added to the gravity of the prognosis.

CLASSIFICATION OF THE TRAUMATIC FORCE

The types of forces that cause injury of abdominal viscera without cutting or perforating the wall are classified into three groups[2]: (1) compression; (2) traction; (3) disruption.

Compression force can be subdivided into sharp, such as a blow, and gradual. These may be local or general. Usually the injuring force compresses the organ against the spinal column. Such a force is capable of causing injury to both solid and hollow viscera and may drive fragments of ribs or pelvic bones into viscera.

Traction force causes injury at or near the junction of fixed and mobile portions of the viscera. Tearing occurs when the

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mobile portion is suddenly arrested by the fixed portion. Veal[3] attributed the mesenteric border tears he encountered to this mechanism. The mesenteries, the duodenojejunal junction, and the ileocecal junction are fixed portions of the intestinal tract.

Disruption force is exerted against the inner walls of hollow viscera, causing the wall to give way. Areas weakened by diseases or previous injuries are more prone to burst than normal wall. Enemata given with too much hydrostatic pressure or given directly from water taps, are common sources of injury. Compression forces applied over hollow viscera in a state of distention will cause pressure with bursting: examples are the stomach shortly after a meal, the bladder distended with urine and the pregnant uterus.

CLINICAL CLASSIFICATION

Estes[4] classifies nonpenetrating trauma of the abdomen into three groups: (1) severe multiple injuries which are rapidly fatal and for which no treatment is of avail; (2) injuries that obviously require immediate operation; (3) cases in which the diagnosis is doubtful and the indications for or against operation are not clear. Most of the patients will be in group 3.

In analyzing fifty-five cases we have found it simpler to classify them according to the nature of the accident into five categories, namely: (1) blows; (2) falls; (3) vehicular accidents; (4) compression injuries; and (5) disruptions. It is obvious that motor vehicle accidents include 1, 2, 4, and 5. Clinically we have divided them into injuries of solid organs, injuries of hollow viscera, injuries of the diaphragm, and retroperitoneal hematomata of undetermined origin.

Fifty-four records reviewed in the paper were taken from the Charity Hospital at New Orleans. One, through the courtesy of Dr. F. L. Cato, was taken from the files of Touro Infirmary. Included in this series are 14 reported by Dr. Isidore Cohn.[5] His commendation of the resident staffs of the three divisions of Charity Hospital is further justified by our study of the additional cases.

Five patients who were injured during the process of birth are included because they showed compression injuries of abdominal viscera. It can be stated that the mildness of the abdominal injuries found at autopsy indicated that they played no part in the death of 3. Two additional cases were included because of compression injuries acquired during the administration of artificial respiration. We have reasons to believe that both of these patients were dead from anoxia before the injuries occurred. All of the injuries occurred during the period from December 31, 1942 to January 1, 1949.

Incidence. There were 484,004 traumatic cases seen in the Accident Room of Charity Hospital during this same interval of time. The percentage incidence of nonpenetrating wounds of the abdomen was the very low 0.011 per cent.

Sex. There were 44 males (80 per cent) and 11 (20 per cent) females. This is not unusual, for males predominate in most of the reported series.

Age. All age groups are represented in this series, from the newborn to 80 years. Nearly one-half of the injuries occurred during the first two decades of life and over two-thirds during the first four decades. Four individuals over 70 received serious nonpenetrating abdominal injuries.

Cause of injury. Motor vehicles played the most important role in causing injury. Of the 55 accidents 26 were caused by them; therefore, nearly one-half can be classed as road casualties. Blows to the lower chest and abdomen accounted for 11 while falls were responsible for 10 injuries. An infant five days old suffered a rupture of the cecum during the administration of an enema. One patient getting artificial respiration received a rupture of the stomach when pressure was applied over that organ while it was distended with anesthetic gases. The other, a poliomyelitis victim with many episodes of respiratory failure, sustained a rupture of the diaphragm while in the respirator.

Time intervals. The average time between acquiring the injury and admission

to the hospital was 13.3 hours. This average is high because in 9 patients concerned they did not seek admission to the hospital until fourteen to ninety-six hours had elapsed. In all 9, the patients were not considered seriously ill during that period. These patients illustrate the necessity for close and constant observation of those who receive blunt trauma to the abdomen.

The average time elapsing between receipt of the injury and operation was 32.8 hours. Besides the late admissions in some cases, other causes for delaying operative treatment were: (1) severe injuries of other parts of the body, especially fractures of the skull, the extremities, and the lower ribs; and (2) mildness of early abdominal symptoms, and the tardy appearance of important symptoms and signs in many. In 2 evidence of the seriousness of the injuries did not become manifest until six and ten days. The first was discharged from a hospital after being observed for twenty-four hours; the second was hospitalized during the entire period because of other injuries.

Mortality. If 5 patients, (3 premature infants and 2 respiratory deaths) are excluded, the mortality for the remaining fifty cases is 42 per cent. Thirty-six patients had abdominal explorations with a mortality rate of 22 per cent. Fourteen were not operated upon and the mortality in this group was 92 per cent. The last figure includes those who died in the emergency room and those who died shortly after admission. Eighteen were operated upon within twelve hours of receiving the injury with a mortality of 16 per cent. Seventeen operated upon after twelve hours had a 23 per cent mortality.

INJURIES OF THE SOLID ORGANS

Injuries of the liver, spleen, and kidneys are more likely to result from sudden blows than from slow compression. Their partial fixation enables them to tolerate slowly applied pressure fairly well.[6] Injuries of the pancreas are rare and are often associated with other serious lesions forming a fatal combination. The liver and spleen are most frequently injured.

Liver. Liver injuries may be classified according to Sheddon and Johnston[7] into three groups: (1) rupture of liver tissue with simple laceration of Glisson's capsule represents minimal trauma; (2) separation of the capsule by subcapsular hemorrhage and damage to underlying structures; and (3) a central rupture with hemorrhage into the parenchyma with later abscess formation.

Small tears will seal themselves off with comparatively little bleeding. This is especially true in liver injuries received in warfare. Rob[8] found that only 1 of 34 cases needed surgery to control hemorrhage. On the other hand, the ruptures caused by nonpenetrating trauma usually result in profuse bleeding and the syndrome present after a short time is one of serious internal hemorrhage.

The early diagnosis of liver rupture is made by the history and the site of trauma, signs of intraperitoneal hemorrhage, localized tenderness, rigidity (has a tendency to spread), and elevation and immobilization of the right leaf of the diaphragm. The bradycardia (50) noted by Finsterer[9] may be present but should not be considered as characteristic of liver injury. It is noted in rupture of duodenal ulcers and in massive hemorrhage not of liver origin. The cause of pulse rates between 50-70 per minute is attributed to vagal stimulation. The blood pressure may be sustained, especially in young individuals with elastic vessels, for a long period, dropping rapidly to zero when death approaches. Small quantities of blood in the abdominal cavity cannot be demonstrated and attempts to percuss shifting dullness[10, 11] are to be condemned rather than encouraged. Patients with intra-abdominal injuries should be kept quiet; movement only adds to shock and tends to start bleeding that has been arrested by clotting. We do not encourage abdominal wall puncture for the diagnosis of hemoperitoneum.

Blood studies are of little help in determining the severity of hemorrhage in the first few hours.[10] We must depend almost entirely on our own observation of the patient and an accurate appraisal of his gen-

eral condition to guide us to the correct diagnosis and the decision to operate or to wait.

Symptoms and signs which appear later are jaundice, peritoneal reaction to blood and bile, abscess formation, and the toxic effects from liver necrosis.

Operation is indicated when there is evidence, or even suspicion, that bleeding is progressing. An adequate incision that will permit complete exploration of the abdominal viscera is imperative. Hemostasis may be difficult to accomplish. Mattress sutures of catgut on atraumatic needles sometimes are sufficient to stop bleeding; however, packing must be resorted to frequently. Long narrow strips of gauze placed against the raw surfaces will arrest hemorrhage and provide for drainage. One of the newer hemostatic agents may be used in conjunction with the pack, although these agents may enhance liver sequestration. Whether packs or sutures are used, *drainage is imperative.* The death rate is extremely high in those patients who are not adequately drained.

Rupture of the liver occurred in 23.6 per cent of the 55 cases. This percentage is at variance with many of the published statistical studies of similar injuries. There were 13 patients with liver injuries. Eight of the 13 had no evidence of injuries of other abdominal organs, although there were intracranial hemorrhages in 2 of the newborn infants. None of the 8 patients survived. The one patient who was operated upon and closed without drainage died on the tenth postoperative day. The remaining 5 patients had multiple injuries of solid viscera. In none was a hollow viscus injured. Three were operated upon: one with rupture of the liver, spleen, and left kidney died one hour after operation; the second with rupture of the liver and one kidney died during the operation; and the third survived after splenectomy and suture of a laceration of the left lobe of the liver. The mortality in the 5 complicated cases was 80 per cent.

Spleen: One would think that because of its location and size the spleen would be invulnerable to injury. Yet it is one of the most frequently injured abdominal organs and has taken precedence over the liver in this group of cases. The architecture of the organ is such that even mild trauma may result in tearing of its soft parenchyma. Unlike small wounds of the liver, once tearing of splenic tissue has occurred, there is a tendency for bleeding or oozing to continue for a long time.

Clarkson[2] divides injuries of the spleen into three groups. In the first group, rupture is followed immediately by massive hemorrhage which rapidly progresses to a fatal issue; the vessels in the main pedicle are torn. In the second group are placed those ruptures with initial hemorrhage and collapse, then recovery. After a latent period, usually hours, hemorrhage again occurs which without splenectomy will result in death. In the third group are placed the subcapsular hematomata. In these the splenic pulp is torn under an intact capsule. These are dangerous; while the bleeding continues and pressure under the capsule increases, there is a period of two to ten or more days when the patient seems to have no serious intra-abdominal injury. When all seems perfectly well within the abdomen the hematoma breaks through the capsule and profuse hemorrhage occurs. We believe that two patients in the series being discussed died from profuse secondary hemorrhage following ruptures of subcapsular hematomata. A third had the spleen removed when a large subcapsular hematoma was found at exploration.

An accurate diagnosis of rupture of the spleen frequently is impossible to make. The nature and site of the injury, combined with pain, tenderness, and rigidity, will direct attention to the abdomen. At times these symptoms predominate in the left upper quadrant, indicating that the spleen most likely has been injured. Added to these are the signs of intra-abdominal hemorrhage; these findings were the only constant factors in our group of cases and indicated the probability of splenic injury. Pain in the left shoulder is mentioned as a very helpful symptom by some authors; we

have found it present only once in 23 cases —the patient had a fracture of the left humerus. Decrease in the respiratory excursion on the left side may be due to rupture of the spleen, fracture of the lower ribs, or both. The possibility of splenic rupture with early or delayed hemorrhage must be borne in mind in all patients with rib fractures of the left side.

Abdominal exploration is indicated when injury of the spleen is suspected. Splenectomy is advised even in patients with hematomata for reasons that have already been stated. Careful examination of the left diaphragm for rupture should invariably be done because the lesions are sometimes associated.

There were 23 ruptured spleens in the series, 41 per cent of 55 patients. Sixteen were the result of vehicular accidents, 6 from falls, and 1 from a blow. Eighteen (81.6 per cent) were not associated with injuries of other abdominal viscera while 6 (18.4 per cent) had associated kidney, liver, urinary bladder and retroperitoneal lesions.

Two patients, aged 60 and 70, had fractured skulls and died without operation. One, aged 79, died from pulmonary edema on the third day without surgical intervention. The fourth death occurred one hour after surgery and was probably due to shock. Splenectomies were done on the remaining 14 patients. The mortality with splenectomy was 6.3 per cent; without, 100 per cent.

Of the group of 5 patients with multiple abdominal injuries, 4 were operated upon with a mortality of 50 per cent. One, not operated upon, died.

The total mortality from rupture of the spleen, including all cases, was 34 per cent.

Kidney: Campbell[12] estimates that the relation of renal trauma to surgical admissions is 1:3,000. He considers hydronephrosis, calculi, and long standing urinary tract infection as predisposing factors. Ninety per cent of kidney injuries occur in the male.

Injury of a kidney by blunt force is recognized more readily than liver and spleen injuries. Hemorrhage from an injured kidney will remain outside of the peritoneal cavity and produce a fullness in the region of the organ. Hematuria will be present unless the ureter is divided or the renal blood supply cut off by division of the pedicle or massive renal thrombosis.[12] Intravenous pyelograms are extremely helpful to determine the degree of renal damage, existence of perirenal extravasation, and the presence or absence and function of the other kidney.

Conservative treatment should be used when there is mild hematuria, no increase in size of the mass in the lumbar region, and the blood count hourly remains stationary.[12] Surgical intervention is indicated when there is evidence of extensive injury to the kidney or ureter, or when there is progressive hemorrhage, or extravasation of urine. Total or partial resection will depend upon the amount of damage found and the presence of another functioning kidney. Adequate drainage should always be provided.

Ruptures of the kidney were found in 9 per cent of the injuries we reviewed. Three cases were not associated with other abdominal injuries. One patient, aged 65, who had a crushing injury of the chest on the opposite side died. One of the 2 that lived was treated conservatively; the other was explored for rupture of the right kidney and was found to have a congenital absence of the left kidney. The mortality was 33.3 per cent.

Two patients who had kidney damage associated with liver and splenic injuries were explored; both died.

Pancreas: Nonpenetrating injuries to the pancreas are uncommon. Shallow and Wagner[13], after a study of their 28 cases and the reported series of acute pancreatitis, determined that trauma plays a part in from 2 to 4 per cent of the cases. Because the pancreas is soft and very vascular, mild force applied anteriorly may cause injury. According to Venable[14] the tear or rupture that occurs may be complete or incomplete, and may divide the duct of Wirsung. Trauma is followed by edematous

or hemorrhagic pancreatitis. The latter usually results in the formation of abscess or pseudocyst. Hemorrhage that follows rupture remains retraperitoneal and forms a mass above the umbilicus.

The diagnosis is made on the following findings: (1) Presence of moderate to very severe pain in the upper abdomen following trauma; (2) development of a mass above the umbilicus; (3) after a short time a large collection of ascitic fluid;[10] (4) a rapid early rise in serum amylase (high normal 180 Semogyi units). Edmondson and Burne[15] found the serum calcium below 9 milligrams per 100 cubic centimeters if there is a hemorrhagic element; values below 7 indicate a fatal outcome.

Treatment by conservative measures should be employed as long as there are signs of clinical improvement. Patients who do not improve but seem to get worse during a six to eight hour period of observation should be explored. Adequate drainage should be established to prevent the spread of peritonitis and the formation of abscesses and pseudocysts.

There were no patients with clinical or necropsy evidence of pancreatitis in our series.

INJURIES OF HOLLOW VISCERA

Immediately following injuries of the trunk it may be impossible to determine definitely the presence or absence of intraperitoneal lesions. The "pseudoperitoneal syndrome" or "traumatic peritonism" of Eliason[17] and Hinton,[18] occurring immediately after injuries of the chest and retroperitoneal area, may be misleading; these injuries very closely simulate intraperitoneal injury. Although confusing at first, the false signs tend to improve early while the true signs and symptoms of peritoneal reaction become worse with time. The presence of air when found by x-ray examination is conclusive evidence that real bowel pathology exists.

The most important findings that point to injury of the gastrointestinal tract are: (1) the history and site of trauma; (2) localized pain, tenderness, and rigidity, which persist and at the same time have a tendency to spread; (3) absence of peristaltic sounds; (4) the quiet attitude of the patient; (5) loss of abdominal respiratory movements; (6) a rising pulse rate; (7) persistent vomiting; (8) the extremely important finding of free air or gas in the peritoneal cavity or in the region of the right kidney; (9) a rapidly increasing leukocytosis, with polymorphonuclear leukocytes predominating; (10) abnormalities of the hematocrit, hemoglobin, red cells, and specific gravity of the blood. Absence of one or more of these does not rule out the existence of serious injury.

It is important that x-ray examination be done with the patient supine, prone, in the right and left lateral, and upright positions if the condition of the patient permits. The presence of free air indicates most likely rupture of the stomach, duodenum, upper jejunum, or colon; the lower jejunum and ileum are less likely to show free gas.

Ruptures of the stomach wall, peritonealized portion of the duodenum, and large tears in the small and large bowel, will show early and progressive signs of acute pathology in the abdominal cavity; so, one can hardly overlook the fact that a grave abdominal emergency exists. There are other injuries, however, that are not as evident and are frequently unsuspected until symptoms and signs of local peritonitis with abscess formation or spreading peritonitis appear days after the injury.

Among the traumatic lesions that present the latent period are: retroperitoneal injuries of the duodenum; small blowouts and tears of the intestine without eversion of the mucosa that seal themselves off temporarily, only to rupture later; and extraperitoneal injuries of the rectal wall.

Lesions that have such a protracted, quiet interim, before the storm, illustrate the inadequacy of a twenty-four hour period of observation. One patient in our series appeared normal until the sixth day following the trauma, when diarrhea occurred; this was followed by secondary rupture of a small diverticulum of the de-

scending colon that had been sealed off by a hematoma. Recovery followed rehospitalization and laparotomy with drainage of the abscess.

Once the diagnosis of ruptured hollow viscus is made, or when it is doubtful because rupture cannot be excluded, surgery should not be delayed. While the urgency for immediate operation is not as great as with intraperitoneal hemorrhage, unnecessary delay adds to the gravity of the situation and increases the mortality. Kornblith[17] cites figures for jejunal injuries which confirm our conviction regarding the folly of procrastination. This author reports that of those who were operated upon within six hours there was a 35-50 per cent mortality, after twelve hours 75 per cent, and beyond that with no operation 93-100 per cent mortality.

Included in our series was 1 rupture of the stomach, which probably occurred post mortem. There were no duodenal injuries. There were 6 ruptures of the jejunum: 1 complete division, treated by end-to-end anastamosis; 1 with two perforations of the wall, two and three feet distant from the ligament of Treitz, with a tear in the mesentery; and 4 others had small lacerations. There was no clinical evidence of shock in any of these patients. Five were operated upon within ten hours, and the sixth, seventeen and a half hours after the injury. There were no deaths; the usual reported mortality is 35 to 50 per cent.

There were 5 traumatic perforations of the ileum. That considerable difficulty is encountered in establishing an early diagnosis in tears of this portion of the intestine is shown in the lateness with which these patients were explored; only 1 was taken to surgery under ten hours from the time of the accident. One was operated upon forty-four hours after acquiring the injury, while another waited ninety-six hours before entering the hospital. The other two patients died twenty-eight and forty-eight hours after injury without operation. Shock was recorded as present in every patient. Generalized peritonitis was a marked feature in all save for the 2 who recovered. The mortality was 60 per cent.

There was one "blow out" of the cecum in a five day old patient, received during the administration of an enema by a mother inexperienced in this procedure. Death followed cecostomy.

The second large bowel injury resulted from a blow to the left lower abdomen. The true nature of the injury was not discovered until six days later when signs of local peritonitis and free air under the diaphragm were found. At operation a rupture of a small diverticulum with abscess formation in an old blood clot was found. The abscess was drained and recovery followed.

Injuries of the urinary bladder are identified by: (1) suprapubic pain; (2) signs of extravasation of urine into the peritoneal cavity or into the tissues of the abdominal wall; (3) hematuria, dysuria, or anuria; (4) abnormalities of the cystogram, using contrast media; and (5) fractures of the pelvis.

Three ruptures of the urinary bladder are recorded. All occurred in automobile accidents and the patients were in severe shock when admitted to the hospital. Two had multiple injuries and died after suprapubic cystostomy. One whose laceration of the bladder was sutured twenty-four hours after injury survived. There were no fractures of the pelvis associated with these injuries. The mortality was 66.6 per cent.

DIAPHRAGM

Ruptures of the diaphragm usually occur as a result of compression being applied to a tense abdominal wall or from puncture and laceration of this structure by indriven fragments of fractured ribs. The injury is suspected because of the dyspnea, cyanosis, pain in the chest, vomiting, peristaltic sounds in the chest, and is confirmed by x-ray examination. Repair should be done early to avoid possible strangulation of the abdominal organs which may become incarcerated in the rent.

There were three injuries of the dia-

phragm in this series, an incidence of 5 per cent. One occurred in a bulbospinal poliomyelitis victim who at the time was in a respirator. One had a fragment of rib driven through the right leaf of the diaphragm and into the liver. The third received a tear of the left leaf and a hematoma of the spleen. Closure was carried out in the third case; the first died from anoxia and the second died from multiple injuries before surgery could be carried out.

MANAGEMENT OF EARLY INJURIES

Serious injuries resulting from nonpenetrating trauma of the abdomen, while not common, occur frequently enough to warrant a definite plan of management. The excessively high mortality is in a large measure the result of careless handling and transportation of patients in shock, incomplete physical examination, and failure to observe patients for a sufficient period after injury. We suggest that the following general plan be employed:

1. Measures to combat shock must be instituted immediately. These include the intravenous administration of glucose-saline solutions and human plasma until whole blood is obtained. The presence and degree of shock is determined by the appearance of the patient. Normal pulse rates and blood pressure readings do not eliminate the presence of shock. The quantity of blood to be administered is determined by the needs of the individual patients; it should be supplied in adequate amounts, and lately we have been using arterial transfusions in the very severely shocked patients.

2. A detailed history should be obtained and a very thorough physical examination made. The important facts that must be ascertained are: (a) the presence and severity of injuries outside of the abdomen, with or without active bleeding; (b) the presence of progressive intraperitoneal hemorrhage.

3. Patients with active intraperitoneal hemorrhage will for the most part follow one of three courses: (a) will not respond to antishock treatment but will show progressive decline to death in about three hours; it is in this group that intra-arterial transfusions may prove to be of great benefit; but our past experience influences our current opinion that death will probably occur, regardless of what is done; (b) will respond to whole blood transfusions but cannot maintain the gain without further administration of blood; (c) will improve rapidly, demonstrating that hemorrhage has been arrested temporarily or permanently. Those patients in group *b* require immediate operation to arrest hemorrhage. Those in *c* require: immediate operation if the spleen is suspected; expectant treatment, if in the opinion of the surgeon, the liver is thought to be the source of the hemorrhage.

4. Hourly blood determinations, ten minute blood pressure, pulse and respiratory recordings should be made. These may or may not presage significant intra-abdominal injury.

5. Patients who show signs of abdominal injury but only mild shock and no evidence of intraperitoneal hemorrhage are not dire operative emergencies. Sufficient time may be taken to have the confirmatory laboratory and x-ray work done. Celiotomy can then be carried out with possibly a more definite diagnosis; if there is still doubt, operate.

6. Patients who present masses in the region of the pancreas will call attention to this organ; in the flank to the kidney or to retroperitoneal hemorrhages not of renal origin. Adequate investigation to ferret out the source of the bleeding is in order and unless the hemorrhage is alarming, which ordinarily it will not be, conservative treatment can be carried out.

7. Very careful consideration must be given to individuals whose first marked symptoms and signs are transient and in whom there is apparent early complete recovery. Since dangerous injuries with latent periods are usually found in this group, these patients should be hospitalized for observation. The length of hospitalization required is variable. Twenty-four hours is the usual time prescribed but this

is entirely inadequate for many; secondary hemorrhage from ruptured hematomata and reopening of perforations of the gut occur occasionally many days after acquisition of the injury. Periodic observation should be made at the patient's home after discharge from the hospital. If possibility of injury of the spleen exists, the patient should not be allowed to leave the hospital. We recommend explorations as the mortality from secondary hemorrhage of this organ is sufficiently high to warrant this procedure.

CONCLUSIONS

1. In this series of 55 cases the spleen was injured more frequently than any other abdominal viscus. Removal of ruptured spleens unassociated with other visceral injuries resulted in a mortality rate of 6.3 per cent.

2. Liver injuries were more frequently associated with other abdominal injuries and resulted in a higher percentage of fatalities. Liver damage occurs more frequently during birth than is suspected.

3. Injuries of the jejunum lend themselves to early diagnosis. They were the most common gastrointestinal tract injury in the series. Early diagnosis and treatment resulted in 100 per cent recoveries.

4. Ruptures of the ileum proved to be more difficult to diagnose and more deadly to the patients.

5. Observation of patients who have had nonpenetrating trauma of the abdomen should extend well beyond the usual twenty-four hours prescribed.

6. We believe that the application of knowledge garnered from others' experiences in dealing with nonpenetrating injuries of the abdomen, consciousness of their occurrence, particularly with relatively mild trauma, their association with other injuries, and the adoption of an orderly plan for investigation and treatment will be of immeasurable help in lowering the present high mortality and preventing the occurrence of disabling remote complications.

REFERENCES

1. Rowlands, R. P., and Turner, P.: Operations of Surgery, 8th Ed., 2:265.
2. Clarkson, P.: Some closed abdominal injuries. Guy's Hosp. Gaz. 56:241 (Nov.) 1942.
3. Veal, J. Ross, and Barnes, E. B.: Rupture of the intestines from non-penetrating injuries of the abdomen. Ann. Dist. Columbia, 10:259, and 281, (July) 1941.
4. Estes, W. L., Jr.: Nonpenetrating trauma of the abdomen. Surg. Gynec. & Obst. 74:419, (Feb.) 1942.
5. Cohn, Isidore: Abdominal trauma from the standpoint of the railway surgeon. Am. J. Surg. 77:162, (Feb.) 1949.
6. Penberthy, G. C. and Benson, C. D.: Injuries to the abdomen in industry, Am. J. Surg. 74:346, (Sept.) 1947.
7. Sheddon, W. M. and Johnston, F.: Traumatic rupture of the liver. New England J. Med. 213:960, (Nov. 14) 1935.
8. Rob, C. G.: The diagnosis of abdominal trauma in warfare. Surg. Gynec. & Obst. 85:147, (Aug.) 1947.
9. Finsterer, H.: Uber Leberverletzungens. Deutsche Ztschr. f. Chir. 118:1, 1912.
10. Katch, W. D.: Diagnosis and treatment of abdominal injuries. Michigan State Med. Soc. J. 42:198, (March) 1943.
11. Noon, Charles: Abdominal injuries arising from road accidents. Practitioner 154:227, (April) 1945.
12. Campbell, F.: Injuries of the kidney. Clin. North America 21:443, (Apr.) 1941.
13. Shallow, T. A. and Wagner, F. B., Jr.: Traumatic pancreatitis. Ann. Surg. 125:66, (Jan.) 1947.
14. Venable, C. S.: Rupture of the pancreas. Surg. Gynec. & Obst. 55:652, (Nov.) 1932.
15. Edmondson, H. A., and Berne, C. J.: Calcium changes in acute pancreatic necrosis. Surg. Gynec. & Obst. 79:240, (Sept.) 1944.
16. Kornblith, B. A.: Rupture of jejunum without evident external trauma. New York State J. Med. 46:2, 1940-1941, (Sept.) 1946.
17. Eliason, E. L. and McLaughlin, C. M., Jr.: Surgical emergencies of the abdomen. Med. Clin. North America 18:63, (July) 1934.
18. Hinton, J. W.: Traumatic lesions of the abdominal viscera. Am. J. Surg. 16:45, (April) 1932.

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PRINCIPLES OF EMERGENCY SPLINTING, TRACTION AND IMMOBILIZATION OF FRACTURES*

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AND
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Seldom is emergency splinting correctly applied, although its value has long been recognized and its use widely recommended. Through the concerted efforts of the pro-

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fession the status of emergency transportation and treatment of the injured has been greatly advanced; however, there is room for further improvement in the early management of fractures. The intent of this paper is to outline the standard principles of early fracture management and to review the improved methods of transportation and immobilization of long bone fractures.

EMERGENCY SPLINTING AND TRANSPORTATION

Following is a summary of standard forms of splinting and transportation to be used at the time of acute traumatic bone injuries of the extremities and of the spine.

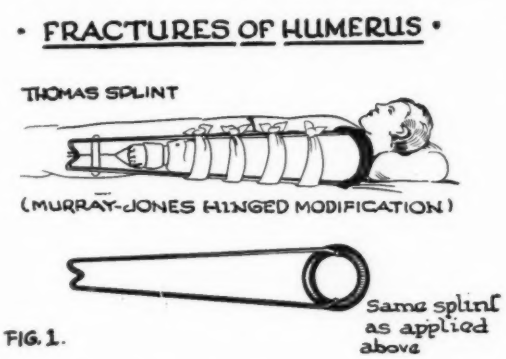

FIG. 1.

The hinged Thomas arm splint (Murray-Jones) (Fig. 1) is most satisfactory for fractures of the shoulder, upper humerus, shaft of the humerus and the elbow. The splint should be applied snugly to the axilla without removing clothing. Traction on the extremity prevents motion at the fracture site thereby lessening the severity of associated pain and/or shock from the injury. An acceptable and more readily available method of splinting for similar fractures is to strap or bandage the arm to the chest wall. (Fig. 2B.) The thoracic cage itself makes an extremely satisfactory splint.

For fractures of the forearm, padded boards are securely strapped on volar and dorsal aspects of the forearm and provide sufficient immobilization for transportation. (Fig. 2 A.) Careful checking of this type of splinting should be made hourly for signs indicative of vascular damage such as swelling, cyanosis, or coldness of the

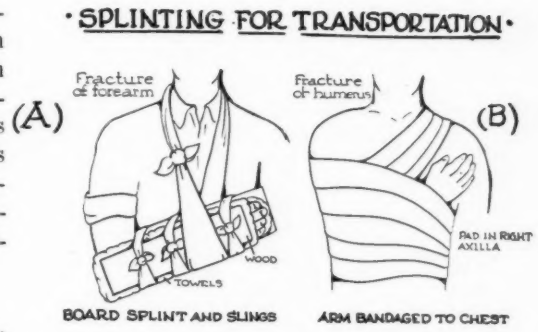

FIG. 2

fingers. Metal splints of various designs have been used for some years and when correctly applied, with subsequent close observation, afford adequate splinting for immediate transportation of fractures of the upper extremity.

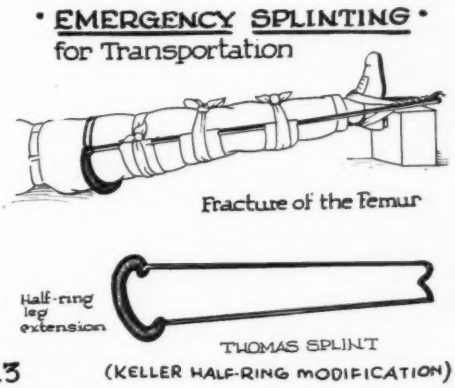

FIG. 3

The Thomas leg splint (Keller half-ring modification) (Fig. 3) is used for splinting and transportation of fractures of the lower extremities above the ankle and the foot. When correctly applied at the site of the injury, with shoe and trousers on, this splint provides traction and lessens the probability of compounding a closed fracture and of deepening the shock usually accompanying a long bone fracture. Thomas splints are made with full rings and with half rings, the latter type being recommended because of ease of storage and application.

Splinting the thigh or leg may also be efficiently accomplished by bandaging the uninjured leg to the injured leg from the

groin to the toes. A board extending from the axilla to the toes may be used for splinting in this region. (Fig. 4 A.) This enables

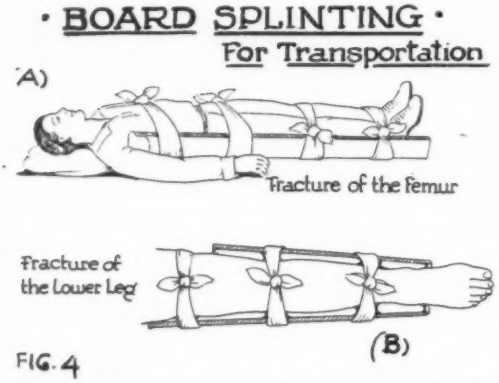

FIG. 4

the patient to be moved as a unit. Board splints on the lateral and medial aspects of the leg are used to splint fractures below the knee. (Fig. 4 B.) Some type of "gutter" splint may be used for transporting fractures of the lower leg and foot. (Fig. 5.) Heavy cardboard is a desirable material to use, for it is firm enough to give immobilization yet malleable enough to conform to the contour of the extremity and to prevent lateral angulation at the fracture site.

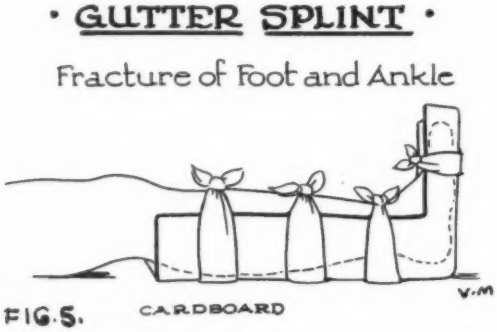

FIG. 5. CARDBOARD

As the majority of spine fractures are flexion injuries, strict adherence to the principles of extension or hyperextension of the spine often prevents further damage to the patient. This may best be demonstrated by two principles, namely: keeping the patient in the prone position while lifting him, and by carrying him face down on a litter or board so that the prone position is maintained until more detailed examination is possible. (Fig. 6.)

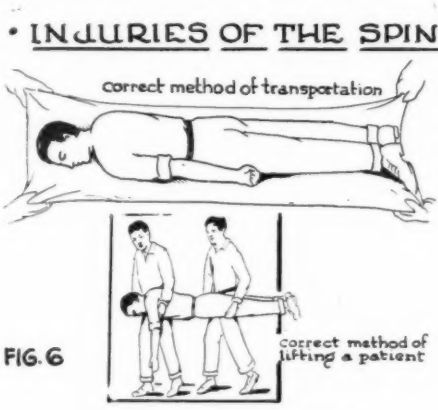

FIG. 6

METHODS OF TRACTION

The reasons for the use of traction in the treatment of fractures are: (1) immobilization; (2) correction of deformity, and (3) restoration of limb length. Skin traction is used when possible. Skeletal traction is employed in cases requiring more weight and pull than can be satisfactorily used on the skin or in cases with damage to large areas of the skin surface.

For the upper extremity, Blake's traction and Dunlop's traction are used. Blake's method (Fig. 7) is commonly employed for injuries of the shoulder girdle and upper

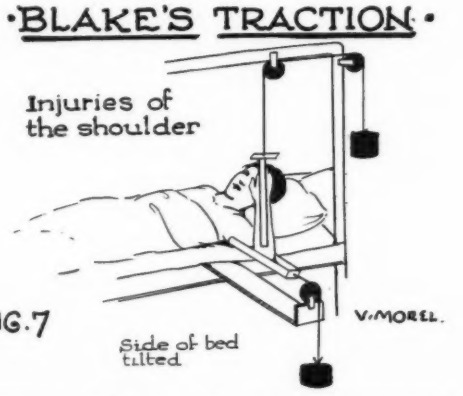

FIG. 7

humerus. After the skin has been prepared with an adhesive substance, such as tincture of benzoin or skin adherent, this form

of traction is applied by the use of strips of moleskin on the lateral and medial surfaces of the arm with lateral traction. To support the forearm another component of the traction is constructed by placing adhesive strips along the anterior and posterior surfaces of the forearm and attaching these to an overhead pulley with sufficient weight to maintain elevation of the forearm. Dunlop's traction (Fig. 8), primarily designed for the reduction of transcondylar and supracondylar fractures in children, consists of skin traction applied to the forearm with lateral traction. The elbow is at 140 degrees and a wide padded sling is placed over the anterior surface of the arm with downward pull of 1 or 2 pounds. The corrective force of posterior displacement of the shaft of the humerus is thereby produced.

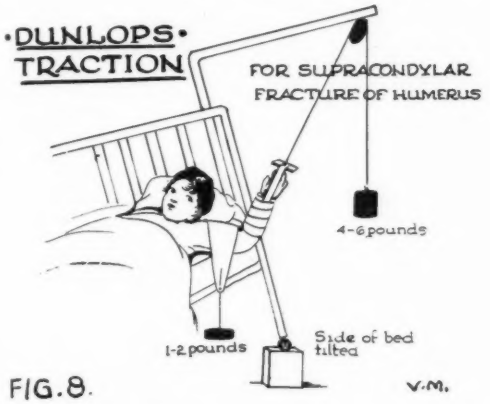

FIG. 8.

Skeletal traction in the upper extremity may be used when needed with Blake's traction. The usual, sites for the insertion of pins (Fig. 9 B) are: (1) proximal end of the shaft of the ulna near the olecranon; (2) the distal end of the shafts of ulna and radius, and (3) distal portion of the shafts of the medial four metacarpal bones. After proper cleansing of the skin surrounding the area, strict aseptic technic should be followed during the insertion of the pins or wires.

Three forms of skin traction are commonly used for the lower extremity. Buck's traction (Fig. 10) is applied from the level of the tibial tubercle inferiorly, employing the same material and method as described in the application of Blake's traction. The amount of pull obtained by Buck's traction is not great but it accomplishes partial immobilization in a fixed position. Care must be taken to avoid pressure on the peroneal nerve as it passes just distal to the head of the fibula. Padding should be applied beneath the tape at this location and also beneath the strips of adhesive as they pass

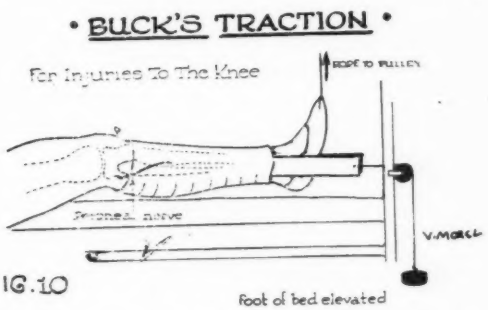

FIG. 10

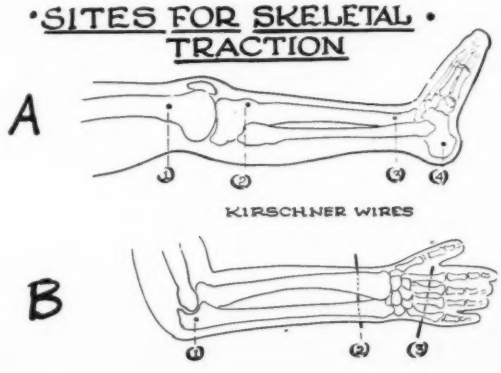

FIG. 9

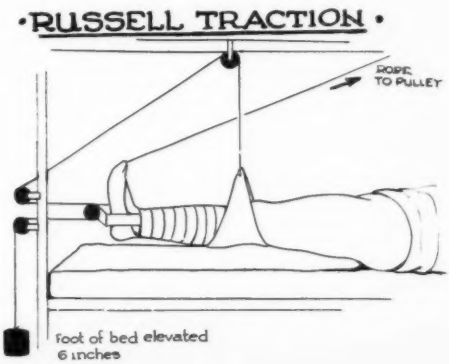

FIG. 11.

over the malleoli of the ankle. Elevation of the foot of the bed on blocks 6 to 8 inches in height will increase the effectiveness of the traction as the weight of the body acts as a force of countertraction. Boards should be placed under the mattresses of all patients in traction of the lower extremities for this allows the leg to glide freely and thereby decreases the amount of friction between the patient and the bed.

Russell's traction (Fig. 11) is applied in a manner similar to that of Buck's method, the difference being in the arrangement of the pulleys with the same amount of weight. A more effective traction results because the pulleys are so arranged as to allow for a compounding of forces which gives, theoretically, twice the pull of the attached weight.

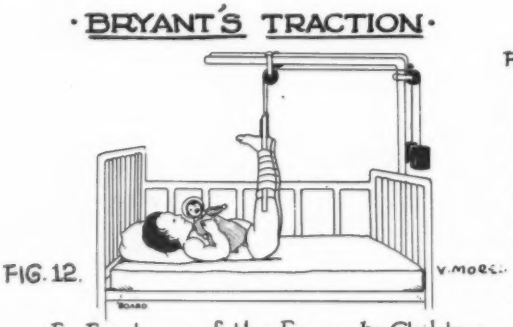

FIG. 12.

For Fractures of the Femur In Children

Bryant's traction (Fig. 12) is a form of skin traction that was originally devised for the treatment of fractures of the femur in children. Adhesive strips are placed along both sides of he leg and thigh from the level of the fracture site distally. Padding is placed over the prominences of the femoral condyles, the peroneal nerve, and the malleoli. Bryant's traction is always applied bilaterally with the legs in complete extension at the knees and ninety degrees flexion at the hips. The pull is directly overhead and the amount of weight necessary to barely lift the buttocks off the bed is the recommended amount, subject to x-ray follow-up at the fracture site.

Skeletal traction is frequently used in the lower extremity. Sites for insertion of wires or pins are illustrated (Fig. 9 A). A Kirschner wire or a 3/32 inch Steinman pin is recommended for the lower end of the femur, upper tibia, and lower tibia. A 5/32 inch Steinman pin is recommended in the relatively soft cancellous bone of the os calcis. For fractures of the femur a balanced skeletal traction is demonstrated (Fig. 13). The thigh and the leg are sus-

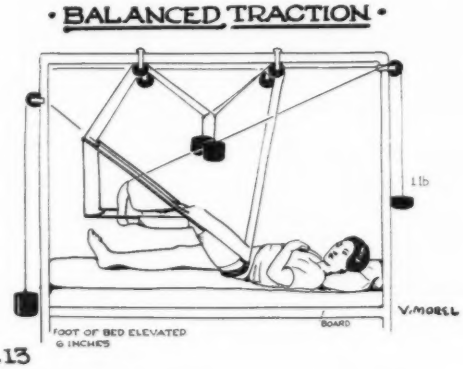

FIG. 13

pended by pulleys to an overhead frame. The traction is maintained in the longitudinal axis of the femur by traction on a wire through the tubercle of the tibia. It is important that the leg below the pin site be elevated with the ankle higher than the knee to insure against dependent edema. A foot piece of some sort is attached so that the ankle is maintained at a right angle. The foot of the bed should always be elevated and a board placed under the mattress.

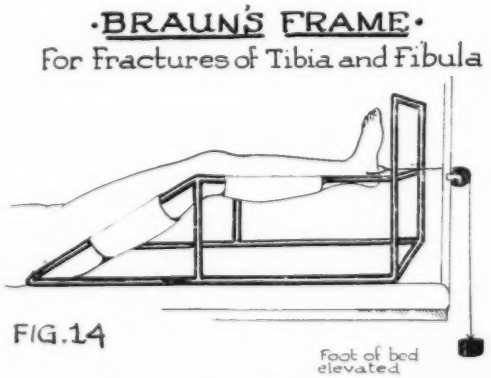

FIG. 14

The Braun frame (Fig. 14) has been used to demonstrate a type of skeletal trac-

tion for treating oblique and or comminuted fractures of the tibia. The pin is placed through the os calcis or the lower end of the tibia and the pull is in line with the long axis of the tibia. The leg and the thigh are suspended on the frame by slings. It is frequently necessary to apply a long padded posterior plaster splint to the extremity so that rotation of the distal fragment at the fracture site is prevented. A hard bed with elevation at the foot is required for this method.

When it is necessary to apply traction to the cervical region, the halter (Fig. 15 A) is commonly employed with a restricted use due to maximum weight tolerated by the patient, 4 or 5 pounds in most cases. For the patient's comfort and for ease in nursing care, it is recommended that two pulleys be used, one attached to each tail of the halter. When necessary to use a small amount of weight over a long period of time, a head band type of traction may be used. (Fig. 15 B.) Made of webbing with adjustable buckle, this band engages the occiput and the frontal bosses for purchase and is also applied with two pulleys. The head band is effective in children who do not easily tolerate the halter. When more effective pull is required there are other forms of skeletal traction which are used successfully. The Crutchfield tongs (Fig. 16 A) have been employed for a number of years and, when correctly applied, serve to maintain traction in the extended position as is desirable. Zygomatic arch hooks (Fig. 16 B) may also be used for skeletal traction and may be fashioned by shaping a small Steinman pin. They are easily inserted in the bedside under local anesthesia. Two pulleys should be used and the amount of weight will prove sufficient to obtain traction for any condition present. By whatever method selected, the head of the bed should be elevated from 6 to 8 inches and the bed should be a firm one. Any desirable amount of hyperextension can be produced and maintained by placing additional mattresses and pillows beneath the upper portion of the trunk of the patient.

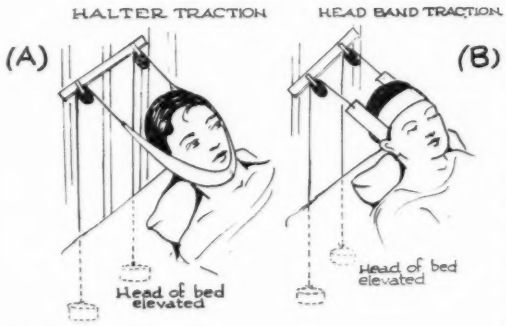

FIG. 15

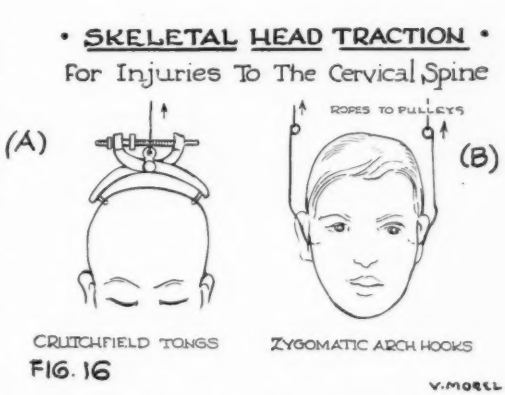

FIG. 16

PLASTER IMMOBILIZATION

We have demonstrated what may be considered as the functional position of the upper extremity, and feel that the following positions should be maintained as often as possible, subject to individual fractures, when plaster of Paris casts are used for immobilization. This includes flexion of the elbow to 90 degrees, midposition of the forearm with respect to pronation and supination, 15 degrees of dorsiflexion at the wrist. If immobilization of the fingers is necessary, partial flexion of metacarpophalangeal and interphalangeal joints should always be employed. Long arm casts should extend from high on the arm inferiorly to the metacarpophalangeal joint on the dorsal surface of the hand and to the proximal palmar crease on the volar surface. (Fig. 17.)

• EXTENT OF LONG ARM CAST •
For Fractures of Radius and Ulna

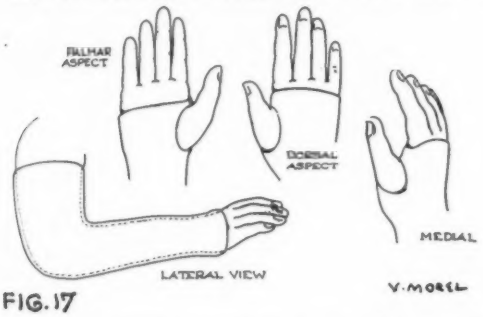

FIG. 17

In the lower extremity (Fig. 18) a long leg case should extend from as high on the thigh as can be comfortably applied, inferiorly to the base of the toes on the dorsum of the foot, and projecting beyond the toes on the plantar surface. Short leg casts should extend from the level of the tibial

• EXTENT OF CASTS IN LEGS •

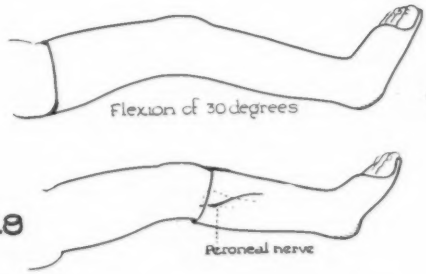

FIG. 18

tubercle to the toes, as mentioned above, with care taken to engage the head of the fibula laterally. Functional position in the lower extremities is accomplished by placing the knee at 150 degrees and the ankle at 90 degrees.

Padding of cotton sheet wadding or stockinette should be used under all casts for fresh fractures, with the addition of squares of felt around and over bony prominences. The use of plaster that is unpadded should be restricted to extremities in which there is an absence of swelling and no immediate possibilty of swelling.

An example of further use of plaster is the hanging arm cast (Fig. 19) recommended for fractures of the shaft of the humerus. With the ring attached at the level of the wrist or slightly below, the weight of the unsupported cast will produce traction and maintain reduction of the fracture. Injuries of the elbow and bones of the forearm should always be primarily immobilized in a long arm cast.

• HANGING CAST •
Fracture of Shaft of the Humerus

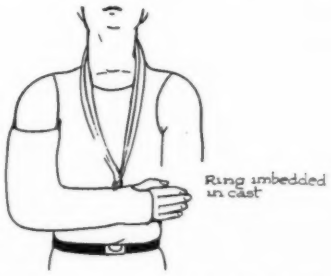

FIG. 19

For immobilization of the femur, a spica and a half cast is necessary in order that the pelvis be fixed. (Fig. 20.) Adequate and positive immobilization of the knee may only be obtained with a spica cast, and

• PLASTER SPICA for FRACTURE of FEMUR •

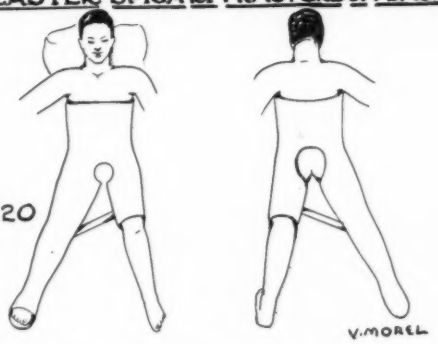

FIG. 20

all fractures of the tibia and fibula, including the ankle, should be immobilized in a long leg cast to prevent rotation at the fracture site. Injuries of the tarsal bones or any area distal to them are immobilized in a short leg cast.

A plaster body jacket (Fig. 21) is demonstrated for immobilization of the lower thoracic and upper four lumbar vertebrae. In order to illustrate the reduction of a

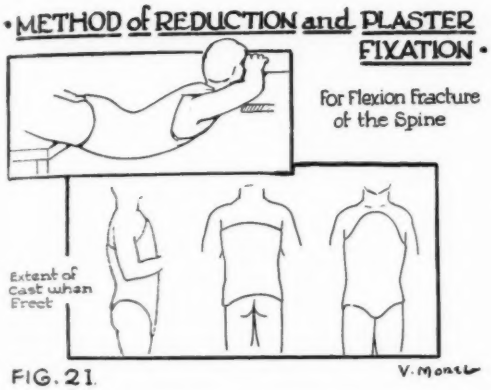

FIG. 21.

compression injury of the upper lumbar vertebrae we have elected to show the postural reduction method of placing the patient suspended between two tables for the application of the body jacket in this position. The extent of the cast anteriorly, laterally, and posteriorly, is demonstrated. (Fig. 21.)

Aftercare of a cast involves a few points worthy of consideration. A wet cast should always be placed on pillows to maintain an even pressure while the plaster is hardening and drying. This prevents the formation of depressions in the cast, which later would cause pressure sores. Elevation of the extremity is very important as the extremity will tend to swell following injury or surgery, and after the application of the cast. Elevation, with active and passive motion of the fingers and toes, will minimize the occurrence of swelling, cyanosis, or coldness—signs of vascular impairment. There should be no pain in the affected part following the application of a cast; however, a complaint of pain should not be lightly dismissed, particularly when the patient definitely localizes the pain. This may be a warning sign of a pressure sore, of necrosis of the skin due to the trauma, or of compounding of the fracture within the cast. Prompt attention and correction may save discomfort or serious complication.

SUMMARY

We have attempted to set forth some of the basic principles associated with the immediate and proper splinting of fractures for transportation; the correct application and uses of the various forms of skin and skeletal traction employed in the treatment of fractures; and the proper extent and padding of plaster casts, with some emphasis on the possible complications frequently resulting from the improper use of plaster, and the prevention thereof.

TRAUMATIC SHOCK*

HUGH C. ILGENFRITZ, M. D.
SHREVEPORT

Peripheral circulatory collapse is the commonest cause of death in patients who have suffered severe traumatic injury. Many individuals who have sustained fractures, lacerations, internal injuries, or even damage to the tissues of the central nervous system following violent trauma may recover without incident; others whose injuries appear to be comparatively minor may succumb to shock within a few hours.

Although many widely different etiologic factors may cause shock, the physiologic basis for the condition and the mechanism of its production still are not entirely clear. The clinical picture, however, is only too well known to every physician.

CLINICAL PICTURE

The patient in shock is characteristically in a state of general collapse; although fully conscious, he may be scarcely able to speak or move. The skin is pallid, cold, and damp, occasionally exhibiting tiny droplets of perspiration. The pulse, at first relatively normal in character, soon becomes weak and thready, often with little acceleration in rate until the later stages of shock. Respirations are shallow, the expenditure of effort by the muscles of respiration being little more than barely sufficient to keep the patient alive. Occasionally a patient in the earlier stages of shock may be restless and excited; frequently also nausea and vomiting may appear. As the condition progresses, however, the excitement gives

*Presented at the Sixty-ninth Annual Meeting of the Louisiana State Medical Society, May 6, 1949.

way to dullness, apathy, and decreased sensitivity, until even the perception of pain may be lost. The blood pressure always falls, often to such a low level that it can scarcely be determined, and the temperature is typically subnormal.

Crushing injuries, especially with fractures of long bones and widespread devitalization of muscle tissue, are most likely to produce shock, which appears an hour or more after injury. Frequently, however, secondary influences such as loss of blood, chilling, insufficient fluid intake, severe pain, fear, or anxiety, may aggravate the effects of a comparatively minor injury and may produce shock unexpectedly many hours after the accident or may increase the severity of a state of shock already developing.

PHYSIOLOGICAL CHARACTERISTICS

While shock can be produced by many different types of injuries such as trauma, burns, or hemorrhage, the physiologic characteristics are much the same. As a result of local injury to the blood vessels following a traumatic accident, plasma and blood are lost into or from the tissues of the traumatized area. This withdrawal of fluid from the blood stream reduces the circulating blood volume, and the cardiac output and velocity of blood flow decrease correspondingly. Reflex peripheral vasoconstriction suffices to maintain the blood pressure above shock level for a short time, but as the vascular fluid loss continues or the compensatory vasoconstriction begins to fail, the blood pressure drops to shock level and tissue anoxia results, with asphyxia and depression of function of the heart, liver, kidneys, lungs, and brain. Hemoconcentration develops as the loss of plasma continues, the erythrocyte count and hemoglobin content of the blood rising in proportion to the withdrawal of plasma. At this stage, the capillaries throughout the body become anoxic and dilated, impounding large quantities of sluggishly moving blood. The increased viscosity of the blood interferes still further with its passage through the capillaries, and the venous return to the heart decreases concomitantly. If the vicious cycle of shock is allowed to persist untreated for too long a time, the tissue asphyxia produces irreversible damage to the vital structures, and therapeutic measures then are of little value.

Traumatic or surgical shock does not appear instantly after the injury has been sustained but appears an hour or more later, since it depends upon a significant loss of blood or plasma. The collapse which follows immediately after a relatively minor injury is often called primary shock, but is actually a transient hypotension due to reflex vasodilatation on a neurogenic basis. Collapse of this type is similar to the sudden drop in blood pressure which may occur during spinal anesthesia or may follow a severe emotional reaction. Here there is a sudden fall in blood pressure as a result of the impounding of a large amount of blood throughout the dilated peripheral vascular bed, but there is no loss of fluid from the blood stream, nor is there any change in the blood volume or the total cell mass. This reaction is temporary and recovery is usually prompt.

Oliguria or anuria may follow traumatic or surgical shock, probably as a result of decreased blood flow in the renal vessels. As a rule, kidney function returns to normal after shock has been controlled by blood transfusion. In some cases, however, especially in the presence of extensive crushing injury, a characteristic renal lesion occurs, with resultant anuria which may prove fatal. The suppression of renal secretion in the crush syndrome is accompanied by necrosis of the distal tubular epithelium in the kidney; this condition, similar to that occurring as a result of severe hemolytic transfusion reaction, has been called "lower nephron nephrosis." While prophylactic measures probably are of little value in preventing development of the condition, it should be foreseen when severe crushing injuries are present. Treatment for shock is instituted as promptly as possible in such cases and, as soon as the patient is in satisfactory condition for operation, all devitalized tissue is excised, fascial compartments are split if the under-

lying muscle appears tense or swollen, and amputation is performed when indicated. It is a wise precaution to chart the daily fluid intake and urinary output in every patient who has suffered a severe traumatic injury, but it is especially advisable when crushing injuries are present.

TREATMENT

Before surgical treatment of a moderately severe traumatic injury is begun, the possible presence of a preshock state should be considered. Even though tissue damage has been relatively severe and a significant amount of vascular fluid loss has occurred, the blood pressure may be maintained within a relatively normal range for a short time by reflex peripheral vasoconstriction. A patient thus may be on the verge of collapse and yet may be able to talk or even sit up. If repair of injuries is attempted before treatment of impending shock is begun, the preshock state may progress suddenly to profound collapse. Premonitory signs of threatened shock include anxiety, agitation, restlessness, pallor, cold clammy skin, rapid soft pulse, and a moderate drop in blood pressure. The reduction in blood volume cannot always be demonstrated early in the course of the posttraumatic state by means of blood counts and hematocrit determinations. Even though enough blood or plasma has been lost following injury to produce a state of shock, the erythrocyte count and the hematocrit value may still be within the normal range for a variable length of time after the accident.

The best treatment of shock is prevention. Many patients who could survive their injuries die of the associated shock. The treatment of fractures, lacerations, head injuries, and even of thoracic and abdominal trauma can wait with safety for one or more hours if necessary until the blood volume has been restored by transfusion, the blood pressure has risen to a safe level, and the patient is no longer in danger of shock. The only two surgical emergencies which offer a greater threat to life than secondary shock are continuing hemorrhage and obstruction of the respiratory tract. Either of these conditions must be relieved instantly; this having been done, the treatment of actual or impending shock takes precedence over the care of any other surgical condition.

The injured patient is disturbed as little as possible; measures are taken to prevent further injury and to reduce discomfort. Fractures are splinted, wounds are covered with sterile dressings, and morphine is administered if needed. Definitive treatment of injuries is postponed until after shock or impending shock has been brought under control. Blankets are used to conserve body heat; hot water bottles or heating pads are not used, to avoid increase of blood flow in the skin and consequent withdrawal of blood from the vital centers. Morphine is given for pain, but never when undiagnosed visceral damage may be present. Stimulant drugs are used only to help maintain the blood pressure at a relatively safe level until transfusions or infusions can be secured. Such drugs are of much value in neurogenic hypotension, when injury has been slight, but are potentially dangerous when the fall in blood pressure is accompanied by significant reduction of the total blood volume.

Restoration of the depleted blood volume is best accomplished by transfusion of whole blood; enough is administered to raise the systolic blood pressure at least to a level above 100 millimeters of mercury. The quantity of blood which may be required varies according to the extent of trauma, the length of time that has elapsed since the injury, the condition of the patient before the accident was sustained, and the amount of blood or plasma which has been lost. In some cases, a state of impending shock can be treated successfully with a single transfusion of 500 cc. of whole blood; in other cases, 40 per cent or more of the total blood volume may have been lost, and from 1,500 to 2,500 cc. of blood may be required for full replacement.

If blood is not immediately available, plasma is the best substitute; it is given rapidly and in sufficient quantity to raise the blood pressure safely above shock level.

No attempt is made to restore the blood volume entirely to normal by transfusion of plasma alone, since the red cell deficit also must be corrected by subsequent administration of whole blood.

If plasma is not available, normal salt solution, with or without dextrose, is given slowly by infusion until compatible blood can be obtained. While less satisfactory than plasma as a substitute for blood, crystalloid solutions are of lifesaving value in supporting the depleted blood volume for a short time.

Other blood substitutes, such as solutions of concentrated human serum albumin and of purified gelatin, appear to be of much value but are not generally available; still others, such as pectin and bovine serum albumin, are efficacious but are unsafe for general use because of their toxic properties.

SUMMARY

Traumatic shock, especially when combined with hemorrhage, is the commonest cause of death following accidental injury. Loss of blood and plasma from the damaged vessels in the traumatized area causes a significent reduction in the circulating blood volume, with a resultant drop in blood pressure and cardiac output below the levels necessary for the maintenance of life. The possible development of shock should be foreseen in any extensive traumatic accident; it can be recognized in its earliest stages by the presence of a persistent reduction in systolic blood pressure. Shock is better prevented than treated once it has developed, since the widespread tissue anoxia caused by the peripheral circulatory collapse may cause irreversible damage to the vital organs within a short time.

Treatment of traumatic shock includes (1) prompt recognition of shock or of a preshock state; (2) transfusion of whole blood as soon as possible, with the use of plasma or normal salt solution as temporary substitutes; (3) use of analgesic and sedative drugs as required to relieve pain and anxiety; (4) prevention of loss of body heat; and (5) avoidance of further injury and postponement of corrective surgical procedures until after shock has been controlled.

DISCUSSION

Dr. C. E. Boyd (Shreveport): I would like to ask why glucose should not be used in head injuries?

Dr. Dean Echols (New Orleans): I have been asked why I lost interest, as most neurosurgeons have, in giving hypertonic solution to people with severe brain injuries. It is true, in almost every neurosurgical center in the United States, that the custom of giving 100 cc. of 50 per cent solution of glucose or sucrose intravenously every four hours to patients with head injuries has been abandoned. The reason for the loss of interest in the once popular "dehydration treatment" is probably the acquisition of the knowledge that the brain is rarely swollen, as previously believed, in severe cerebral injuries. Also, as pointed out in the paper, when spinal punctures are performed on patients with head injuries, the surgeon invariably discovers that the pressure is normal or subnormal. Extensive experimental work on animals and humans in recent years has confirmed this clinical observation. These investigations have shown that there is no cerebral edema of any great consequence in the absence of gross hemorrhage. Therefore, almost everyone agrees now that it is important, not to withhold fluid from the patient, but to give him an almost normal amount from the beginning.

Dr. C. E. Boyd (Shreveport): The reason for that question is that in 1935 at the Charity Hospital in Shreveport some one found that the mortality of head injuries was much lower in Charity Hospital than when neurosurgeons attacked them earlier. That perhaps led to that treatment in vogue here. Since then I read Dr. Echols' article and it is hard to realize or to accept that in severe brain injury without hemorrhage there should be decrease in pressure. I accept his presentation, however.

Dr. Isidore Cohn (New Orleans): In the first place I am particularly happy over this symposium as it is the first time that I know of that the surgical program in the State Society has been devoted to the subject of trauma. One particular reason that I am interested is because, through the efforts of the College of Surgeons' Committee on Fractures and Other Traumas, we are trying to stimulate interest in early adequate handling of trauma. I was so happy over Dr. Echols' paper that I asked if he would give me a copy so that it might be distributed to the members of the committee on trauma throughout the country. With few exceptions he discussed the subject from the standpoint of fundamentals. He has given us a great deal to think about.

I was particularly impressed by the presentation

of Dr. Miangolarra because a little over a year ago I worked with one of the residents at Charity, Dr. Waters, on this same subject.

The important thing is for us to be conscious of the fact that when an individual has a non-penetrating injury, such as a fall or blow on the abdomen, we have to be conscious of the possibility of rupture of the spleen and damage to the liver or damage to the hollow viscera. If we continue, as some do, to see these individuals with a non-penetrating abdominal trauma and send them home, in twenty-four hours or less time, then they come back in a state of shock. My first interest was gained in the old Shreveport Charity Hospital in 1905 when a man was injured by the tail-gate of an express wagon shoving him up against the wall of the railroad station. That man walked to the hospital and did not complain of pain. The house surgeon and interne saw him and nothing was said or done about it and in about four hours the man's abdomen became rigid and autopsy showed it was full of blood and the liver was torn to pieces.

Rupture of the diaphragm, liver, or spleen is not going to be found unless we are conscious of the possibility, and we are going to save a great many more of these people if we do not say "go on home and go to bed," but put them in a hospital where they can be observed carefully and do something about the condition as quickly as possible.

I was particularly impressed by the simplicity of the paper by Drs. Alldredge and Banks. I think that kind of thing is something we all should know more about and do more about.

Dr. Ilgenfritz' paper, the question of management of shock, should impress all of us with the importance of using whole blood and not substitutes. Whole blood is the thing and I think blood bank or catastrophe management should be started. The sooner we become interested in that the more patients we will be able to save.

Dr. J. Q. Graves (Monroe): I was deeply impressed with the idea brought out by Dr. Echols regarding the use of 50 cc. of 50 per cent glucose in the treatment of patients who have sustained head injuries.

The theory of brain injuries in the past, was that edema is associated with the injury, and that concentrated glucose given intravenously would be of benefit, in that it would produce a dehydration. It is refreshing to know that fluids can be given freely, intravenously, without fear of increasing brain pressure.

When complications, such as coma, mild or deep coma, follow injuries of the brain, we now know, they are not due to edema, but caused by a hemorrhage. If the coma deepens and the reflexes are destroyed, surgery is to be thought of. With x-ray and clinical study, localization of the hemorrhage will be determined. If there is evidence of continued hemorrhage, an effort to locate and ligate the bleeding vessel is indicated, and at the same time, to remove all blood clots.

Your symposium regarding traumatic injuries to the abdomen is the opening up of an almost forgotten subject, one which is often presented to the industrial surgeon. Abdominal injuries should be studied carefully for several hours, or until sufficient time for all complications to have developed before one is considered out of potential danger. It is best done in an institution. In some instances, relatively insignificant abdominal trauma can be responsible for an abdominal catastrophe or a tragedy. A full stomach or bowel, under favorable conditions, can be easily ruptured. The spleen and liver are two of the most vulnerable organs of the abdominal cavity. Both are easily damaged, and at times, to the extent of death. In all abdominal injuries careful study should be made of pulse, respiration, temperature and blood pressure reading. If they are followed closely, early signs of impending danger can be detected. If the clinical picture presents sufficient evidence for surgical interference, an exploratory should be done early.

Dr. John G. Snelling (Monroe): As Dr. Cohn brought to our attention, the College of Surgeons is extremely interested in trying to spread the gospel of proper handling of traumatic injuries and it is certainly a fine doctrine to get over.

In so many of these injuries, as brought out this morning the first aid treatment is important, and that concerns, therefore, the general practitioner. I wish there were more of the general practitioners here this morning at this surgical symposium. I think really this thing should reach the general practitioners as far as possible, for they handle the case first. In addition I would like to say that the transportation and proper handling of these injured people by the ambulance crew, and/or laymen who first see the case, is also very important. If we keep preaching this we are going to spread enough of the gospel around to finally have the situation to the point where it is not perfect but much improved over what it has been.

Of course I am particularly more concerned with abdominal work, and thoracic to some extent, but as Dr. Echols brought out the general surgeon still has to handle head injuries of certain types, so he should be interested in all trauma. I have personally seen a couple of cases of delayed rupture of the spleen and liver and think it is highly important that the period of observation for people with any traumatic injury should be carried out meticulously. We should not get out of touch with these patients until we are absolutely sure that any chance of latent or dormant conditions manifesting themselves later, has well passed.

Dr. Branch Aymond (New Orleans): I am interested in the phase of vitamin K as to the part it plays in capillary oozing, and for this reason I would like to know if vitamin K would or would not be of benefit in the treatment of surgical shock, for instance, to include it in the infusion while administering same to the patient?

Dr. Hugh C. Ilgenfritz (Shreveport): I believe that in a patient whose blood prothrombin is considerably below normal, leakage of blood from damaged vessels would certainly be increased. If shock occurs in a patient who might already have a prothrombin deficiency for some reason, such as liver disease, poor diet, pregnancy, and so on, the use of vitamin K would certainly be worth while. When the blood prothrombin is normal, however, administration of vitamin K is not indicated.

Dr. M. B. Pearce (Alexandria): We see a good deal of traumatic injuries around Alexandria. We do not have the benefit there of specialists like Dean Echols to call on but occasionally we send Dean a case we can not handle as we would like to. This symposium on traumatic surgery has been most interesting to me. It is up to us who see the injuries early to take care of these patients. Some of you would not believe it if I would tell you of our experiences particularly in trauma of the spleen. Recently we had occasion to look up a few of our cases in traumatic rupture of the spleen, and believe it or not, we have not had a single death in some 18 cases. One of the last cases we had was a man some forty-seven years of age who had fallen on the ice; surgery was done forty-eight hours later. This patient was admitted to the hospital and x-rayed and found to have fracture of the ribs. The patient went home. The following day he was readmitted to the hospital. The family physician did not realize the fact of a ruptured spleen. I think the original injury did not have the hemorrhage we saw later. When I first saw him he was in shock about two o'clock in the morning and we had to give him two and a half pints of blood before we got him anywhere near a safety level. Even this individual at his age made a satisfactory recovery following splenectomy.

Recently, we have had a number of cases in children. One was a young boy eight years of age who was riding a horse. He pulled the horse up suddenly and the girth broke and he fell. The mother called the family physician and he told the mother to observe him for a while and if he did not get along all right to let him know. The child was pale when the mother called; she made no other comment. About three o'clock in the afternoon she called me and told me the child was pale and told what had happened. I advised her to bring the child in immediately to the office. I examined the child and realized immediately there was intra-abdominal injury. The child was sent to the hospital and splenectomy done and there was uneventful recovery. Rupture of the spleen is the most common intra-abdominal injury and we should always be looking for it and always remember that the spleen ruptures very easily. We have had patients we were examining and thinking of rupture of the liver and find rupture of the spleen. We can not always make a correct diagnosis of rupture of the spleen. In three cases we thought of rupture of the liver and they had rupture of the spleen. Other injuries, particularly of the stomach and pancreas, we have found in a number of cases. Recently, we had a case of rupture of the pancreas with rupture of the duct and thought we were going to get continued fistula. It is impossible, of course, to do anything more than simple suture of the pancreas and one man did drain for quite a while. Later on I asked one of our surgeons what to do for continued drainage. He said just watch a little longer. We did and the fistula closed and there was no more trouble. We do see injury to the pancreas and I think when we explore a patient we certainly should explore the pancreatic region.

With the increased number of automobiles, there are more and more people getting in accidents and we are going to get more and more abdominal, head, and traumatic injuries of all types. I try to make it a rule to hospitalize every individual with head injury of any kind, whether unconsciousness occurred at the time of injury or not. Where possible I think all should be hospitalized and watched carefully for the first twenty-four or forty-eight hours. Many will be hospitalized needlessly but I do not think you will be doing the wrong thing. You can discharge the next day but I believe our constant observation of these cases is well warranted and the patient is entitled to it.

Dr. Gerald N. Weiss (New Orleans): I would like to know what Dr. Ilgenfritz thinks of the work of Dr. Shorr at the University of Cornell in connection with studies on vasotropic factors in experimental shock.

Dr. Hugh C. Ilgenfritz (Shreveport): I am sorry. I am not familiar with the work.

Dr. Weiss: I was interested in knowing what you thought of this work of Ephriam Shorr which was reported in the September 1947 issue of the *Transactions of the Association of American Physicians*. Shorr and his co-workers reported on their findings of vasotropic factors (a vaso-excitor material, VEM, and a vaso-depressor material, VDM) concerned with experimental traumatic and hemorrhagic shock in dogs. The vaso-excitor material in this humoral theory of shock also borders on the explanation of renal hypertension, which aspect is given consideration in the article referred to.

The principle is that shock, hemorrhagic or traumatic in nature, involves humoral factors

which regulate peripheral circulation. The distinction between reversible shock (during which time VEM predominates) and irreversible shock (during which time VDM predominates) is made clear. The VEM, produced by the relatively anoxic kidney, produces a vasoconstriction of precapillary sphincters of the capillary bed, conserving circulating blood and allowing for a "preferential circulation" during the reversible stage of shock. This stage can, of course, be corrected by transfusions and aided at times by adrenalin injection. The VDM, produced by the relatively ischemic liver and the ischemic skeletal musculature, causes a circulatory collapse with generalized capillary vasodilitation, and irreversible shock ensues. The "neutralization" and destruction of VDM was studied, pointing out that correction of "irreversible" shock may be possible. The subject was and is still being studied in great detail. It is actually another humoral theory of shock, one which you did not mention in your presentation. I was interested in your comments on this aspect of the subject.

Dr. Hugh C. Ilgenfritz (Shreveport): I am not familiar with the work; however the humoral idea of shock is one of the oldest of theories. Beginning with the work of Cannon during World War I, extensive studies have been made on the possible humoral mechanisms in the physiology of shock, and each successive report arouses further interest and controversy. I am sorry that I am not familiar with the work described and therefore cannot comment.

Dr. Monroe Wolf (New Orleans): In trauma of the kidney it was said you may not get bleeding. I have seen many cases at Charity and I have not seen one yet without microscopic blood. They are usually in considerable pain and shock when they come in. Usually there is tenderness on the side involved. Unless shock is extreme we give palliative treatment. We had three cases we nephrectomized; one a child three years old. He developed fever and we went in and found the whole upper kidney severed. I assisted the resident in doing the work. He had the severed section of the kidney in his hand and thought he had the pedicle. I had to assist in locating the pedicle.

In trauma of the bladder as well as urethra my policy is to do as little manipulation with catheter and so on as possible. I think you should open the belly in cases of rupture of the bladder. We have had recently a child about ten years old, a boy struck by a truck with a pelvic fracture, thigh fracture and rupture of the urethra. The resident took care of him. Fortunately he found communication of the urethral tear through the suprapubic cystostomy by passing retrograde sounds and inserted a catheter. He neglected to make counter incision in the perineum and drainage had to be done several days later.

As mentioned, drainage should be instituted at the time of surgery in case of extravasation, so that there will be no retroperitoneal infection from extravasated blood which may easily ascend retroperitoneally.

Dr. Dean Echols (New Orleans): I have the uncomfortable feeling that I have disappointed some of you in my indifference toward dehydrating medication. I would like to point out that I did not say that the intravenous administration of a 50 per cent solution of glucose should be avoided; I even mentioned indications for it. Perhaps I can summarize my viewpoint in this way. There certainly is no objection to giving 50 or 100 cc. of a 50 per cent solution of glucose intravenously, if one is confident that there is cerebral edema and increased pressure. However, the point is that the patient probably has cerebral edema and increased intracranial pressure because he has anoxemia. It was formerly believed that injury to the brain caused the swelling and that dehydration was the only course of action to overcome it. Now it is known that the cause of cerebral edema is not the initial injury but the anoxemia that follows, which is due to an imperfect airway. Therefore, a much more effective way to overcome the intracranial pressure is to improve oxygenation. If the patient has labored, noisy respiration, anoxemia is slowly and steadily developing. Thus, a tracheotomy, which relieves the anoxemia, also reduces the cerebral edema.

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MODERN ADVANCES IN DIABETES MELLITUS

FRANK W. PICKELL, M. D.

BATON ROUGE

We, as practitioners, realize today that there are more diabetics to treat, since 4 per cent or more of our present population has now, or is destined to develop the diabetic state, three fourths of them after the age of 50. We also realize that no longer should we assume that they work out their destiny alone, or just with the help of insulin. We believe that they should be aggressively and rigidly regulated, as the patients who most closely control their disease are freer of complications and live longer. The increase in life expectancy, of less than three years in the juvenile diabetic in preinsulin days to over forty years now, should

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be transmitted to the much more numerous middle aged and elderly diabetics who recent statistics have shown have had an increase in life expectancy of only three to four years since the advent of insulin.

Our purpose in this paper shall be to discuss some of the more recent ideas about the physiological upset in diabetes mellitus and then some of the newer and more refined methods in its management. We believe better diabetic treatment is achieved by knowing all new advances and then with caution adapting them to our patients.

Though there continue two schools of thought concerning the nature of the disturbance in carbohydrate metabolism in diabetes mellitus, more and more evidence accumulates to emphasize that under-utilization of glucose constitutes the basic disturbance and that the glands of internal secretion and the liver are also intimately involved. Figure 1 represents very diagrammatically normal carbohydrate metabolism. The blood glucose may be pictured as arising from three types of processes. The first of these is the absorption of glucose across the intestinal mucosa from the diet. A second source, termed glycogenolysis, is from the breakdown of glycogen in the liver. This process has been clarified by the Coris of St. Louis, who have shown that the initial process is one of phosphorolysis, and that its reaction product glucose-1-phosphate is readily transformed in the body to glucose-6-phosphate, and that these two reactions apparently occur wherever one finds glycogen. The next reaction glucose-6-phosphate phosphatase glucose + inorganic phosphate is, in the mammal, more or less peculiar to the liver, although it can undoubtedly occur in other tissues.[10] The lack of phosphatase in muscle is the reason muscle glycogen is not available as a direct source of blood glucose.

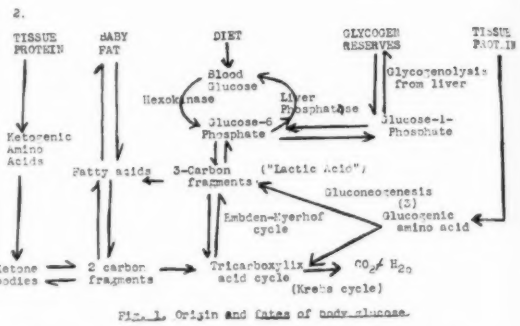

Fig. 1. Origin and fates of body glucose.

Reaction 1. Glucose-6-Phosphate *Phosphatose,* Glucose inorganic phosphate.

Reaction 2. Glucose Adenosine Triphosphate Hexokinase, Glucose-6-phosphate Adenosine diphosphate.

A third source of blood glucose is gluconeogenesis from noncarbohydrate precursors in certain amino-acids, fatty acids, glycerol, and fragments resulting from the breakdown of glucose itself. The relative amount arising from each source in man is unknown, but in experimental animals 3 per cent from glycogen of the liver and 30 per cent from smaller fragments was shown to be contributed as compared to that ingested from diet. Glucose, thus, arises more or less continuously and all of it must be disposed of. It must first be phosphorylated, and the enzyme hexikinase is necessary for this reaction, whcih very likely occurs at the cell membrane. After this more or less obligatory first reaction, the product glucose-6-phosphate, as you see in Figure 1, is exposed to many possible fates. It may be converted to fatty acid, or it may be transformed to the glycogen stores, or finally, it may be oxidized. The amount going in each direction in man, again is all unknown, but in the experimental animal, the rat, 3 per cent of all glucose made or ingested went to glycogen and 30 per cent to fatty acids, and the remainder was metabolized to [8, 10] CO_2 and other products not shown in Figure 1. If this reaction is going normally, one would find normal amounts of glucose converted to CO_2, to glycogen, and to fatty acids each day. Figure 2 indicates the possible magnitude of this, by the thickness of the ar-

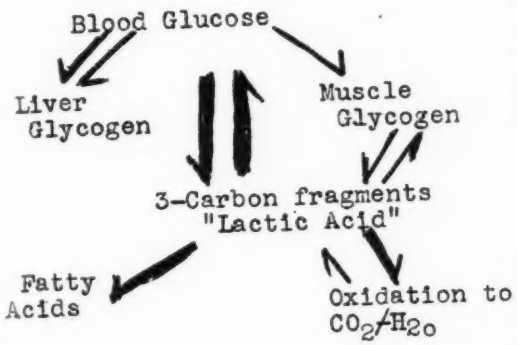

Fig. 2
Normal

rows. In this reaction, in which the abundant enzyme hexokinase is inhibited, one would predict a retardation in all the various modes of utilization of glucose, and it would appear at the present time that this is the situation in the diabetic individual[8, 10]. This situation in diabetes is shown in Figure 3, which should be compared to Figure 2, remembering that the thickness of the

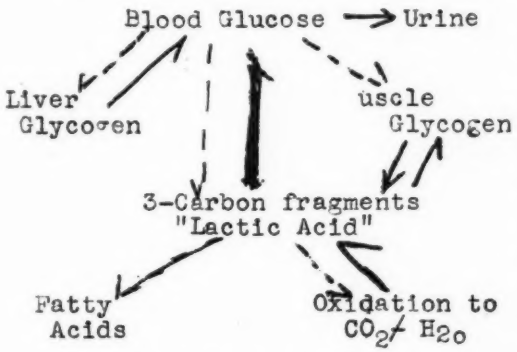

Fig. 3 Diabetes Mellitus

arrows shows the possible magnitude of the reaction. The Coris have shown a specific inhibitor to this reaction can be isolated from the anterior pituitary gland and that its action can be prolonged by certain of the oxygenated adrenocortical steroids. But perhaps their most important discovery was their demonstration that this inhibition of the hexokinase reaction by a specific inhibitor of the anterior pituitary gland is completely abolished by insulin. Normally, a proper balance is maintained and the blood sugar level remains at the normal level of about 100 mgm. per cent. This assigns insulin a satisfactory position as a rate determiner in carbohydrate metabolism. Its over-all effect in magnitude

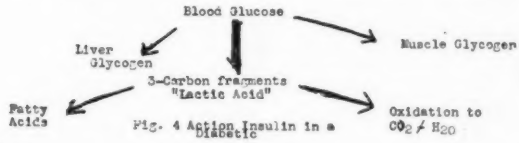

Fig. 4 Action Insulin in a Diabetic

in a diabetic is shown is Figure 4 by the thickness of the arrows. Also it aids indirectly in the synthesis of certain amino-acids used in the elaboration of body protein[10].

A brief look now at the new ideas of the influence of some of the glands of internal secretion on carbohydrate metabolism. The adrenal gland is important. We all know that epinephrine characteristically raises the blood sugar by increasing the breakdown of liver glycogen and later decreases muscle glycogen which breaks down to lactic acid to be transported to the liver for synthesis into glucose or glycogen. Cori suggests that some adrenal steroids exert their "anti-insulin" effect primarily by intensifying the inhibitory action of A. P. E. upon hexokinase[8]. More recently an S-Hormone (sugar hormone) of the adrenal cortex is believed to direct conversion of protein to glucose. This will explain the development of diabetes after moments of chronic stress and also perhaps explains the glycosuria following coronary thrombosis and after emotional upheavals.

Hyperthyroidism in a diabetic has a worsening effect. The thyroid hormone probably acts similarly to the anterior pituitary. Hypothyroidism causes the diabetic state to become much milder.

The pancreas now deserves a word. Over two decades ago, after Banting and Best isolated insulin and firmly established that it was elaborated by the beta cells of its islands of Langerhans and showed it corrected all the obvious defects of carbohydrate metabolism in pancreatectomized dogs and human beings with diabetes mellitus, a simple hypothesis seemed to explain dia-

betes mellitus, but Houssay of Buenos Aires, in 1930, with some startling experiments shook our complacence and gave impressive support to earlier clinical observations in a variety of endocrine disorders that in the pancreas was not the primary cause of diabetes mellitus. This investigator demonstrated that if the anterior pituitary were removed before or after the pancreatectomy, the diabetes resulting was very mild. A simlar ameliorating influence was demonstrated by Lukens to follow adrenolectomy in the pancreatectomized cat. Four reported human cases of the Houssay phenomenon and sixteen cases of coincidence of diabetes and Addison's disease[11] corroborate this experimental work. A recent reported case[9] of total pancreatectomy in a patient with diabetes showed no change in an insulin requirement of about 40 units daily.

Now a word about ketosis. No longer do we say that "fat burns in the flame of carbohydrate" and no longer do we consider fat metabolism any different, qualitatively, in the diabetic. Referring to Figures 1 and 3, let us consider what happens when a diabetic is given a large amount of carbohydrate. This ordinarily should be stored in the liver, but is not, and when glycogen storage is insufficient, then fatty acid production is greatly increased, above what the muscles and tissues can utilize. Here is the reason: At all times there is a constant competition between the fatty acids and stored glycogen for a single enzyme system. Glycogen gets the preference but when it fails fatty acids are metabolized in still more excess. Their accumulation in the blood causes a disturbance in acid base balance and produces an intoxication that is ketosis and it plays a considerable part in the picture of diabetic coma.

The factor of obesity in diabetes is important, as more than 50 per cent of diabetics are obese and a majority of the older age diabetic patients lose evidence of a disturbance in carbohydrate metabolism with a reduction in weight. Allen found that in partially pancreatectomized dogs, showing no evidence of experimental diabetes, a diabetic state could be produced by overfeeding, and after withdrawal of too much food they would return to a normal state.

A family history of diabetes can be obtained in at least 25 per cent of diabetics. In identical twins the evidence of diabetes is 70 per cent in contrast to 10 per cent in twins derived from separate ova. Genetic studies by Joslin and others, strongly suggest that diabetes is inherited as a mendelian recessive trait. A new fourth method for producing diabetes in the experimental animals has been added to the now classical ones of:

1. Pancreatectomy, total or partial with overfeeding.

2. Injection of large amounts of A P E (or in suitably prepared animals, of adrenal cortex or thyroid).

3. Injection of alloxin.

This new method was accomplished by producing a state of hyperglycemia for two weeks in a cat by administering glucose solution intraperitoneally.[1]

The commonest pathological lesions in diabetes mellitus are found in the pancreas, but they are inconstant and absent entirely in 10 per cent of large series autopsied[8]. Intercapillary glomerulosclerosis was found in 44 per cent of diabetic autopsies in one series[8] but only 19.5 per cent in another at Mayo Clinic[13]. Arteriosclerotic lesions are extraordinarily prevalent in patients with diabetes, even in children, as in 50 per cent of children who have had the disease fifteen years, x-ray will show calcification of the arteries. A recent study of 50 autopsied diabetic hearts in Boston, showed 75 per cent had significant coronary artery disease of the arterisclerotic type.[12]

Adequate diabetic treatment aims in general to maintain the patient in a state of well being so that he will avoid complications and degenerative diseases, as evidenced by progressive loss of vision, gangrene, or flagging heart, which may suddenly appear ten or twenty years hence. In all these complications there is just one single common finding—hyperglycemia,—

so the greatest need in the prevention of later diabetic degenerative disease is good control of hyperglycemia at its onset and thereafter. First the diabetic should be put on a satisfying and satisfactory diet and instructed in its adjustment.

A SAMPLE DIET (STANDARD)[15]

TABLE 1

WITH USUAL CALCULATIONS THAT SHOULD BE TAUGHT

	C	P	F
Breakfast:			
Fruit—10%—1 portion	10	1	0
Cereal—½ cup cooked or 2/3 cup dry	20	4	0
Eggs, 2	....	12	12
Bacon, 3 strips crisp	....	8	16
Bread, 1 slice or 2 biscuits	16	3	....
Butter, 1 pat	....	....	7
Milk, ½ cup	4	2	3
Coffee or Tea	....	....	....
	50	30	40
Lunch:			
Meat, Fish, Fowl, Cheese, average portion	....	18	13
5% vegetables as many as desired	5	1	1
10% vegetables 4-6 rounded tablespoons or 20% vegetables 2-3 rounded tablespoons	10	2	....
Bread, 1 slice	16	3	....
Butter	....	....	7
Fruit 10%	10	1	....
Coffee or Tea	....	....	....
	40	25	20
Dinner same as lunch	40	25	20
Total grams	130	80	80
@	x4	x4	x9
Calories	520	320	720
Total calories			1560
To above may be added:			
3 slices bread	48	9	....
3 glasses milk	36	12	18
Total grams	84	21	18
	x4	x4	x9
	336	84	162
Total Calories			582

After the patient has been on such a standard diet for a week, then think of insulin if there is still glycosuria.

Instruct the diabetic to examine the urine for sugar four times daily, 7 A. M., 11 A. M., 5 P. M., and 9 P. M.; that is, before each meal and at bedtime. The early A. M. test reflects the fasting state overnight or the fasting state plus the effects of protamine zinc insulin, if it is being used. The latter three tests reflect the previous meal and its utilization, plus exercise, plus effects of whatever insulin used. The patient should keep a record in a notebook, that can be easily analyzed and the following simple tabular scheme, I find best:

TABLE 2

	7 A.M.	11 A.M.	5 P.M.	9 P.M.	Remarks
Monday	Blue	Green	Blue	Green	
Tuesday	Green	Blue	Blue	Blue	
Wednesday	Blue	Orange	Green	Green	
Thursday	Green	Blue	Green	Blue	
Friday					
Saturday					
Sunday					
Monday					

The color of the sugar test should be written down each time. Use Benedict's solution or Clinitest.

If glycosuria is present, regular insulin twice a day before breakfast and before supper may first be tried, divided as follows: 12 before breakfast and 8 before supper or 16 before breakfast and 10 before supper. This should be gradually increased until the four specimens of urine are sugar free. An alternative start may be made with 10 to 20 or up to 40 units of protamine zinc insulin, daily half hour before breakfast, testing the urine at the same four times and increasing the minimum starting dose until the urine is sugar free.

A great majority of the milder cases may be successfully kept sugar free on the protamine zinc insulin, but these are largely in the older age groups. The objection to protamine zinc insulin is that that it may cause hypoglycemic shock during exercise or during the early hours of the morning. Right now let us look at Table 3[16], and get fixed in our minds the time activity of a single large dose of the various types of insulin, in a moderately severe diabetic, for these are the overwhelming majority of the ones we want to improve the treatment for:

TABLE 3

Type of Insulin	Action Demonstrable in	Peak Action in	Intensity at peak	Duration of Effect:
Regular or Crystalline Insulin	1 hour	3-6 hours	Strong	8-12 hours
Globin	2 hours	8-12 hours	Fairly Strong	24 hours at most
2:1 mixed of regular and P Z I	4 hours	12-16 hours	Moderate	1½-2 days
P Z I	4-8 hours	24-32 hours	Weak	3 or more days

Regular or crystalline insulin is used in the following cases:

1. Emergency infection.
2. To supplement before each meal the "depot" insulin given before breakfast.
3. In the rare cases of high insulin requirement of 150 units or more.
4. To get better control in some cases. Globin insulin tends to give late afternoon hypoglycemic shock and glycosuria at night and we consider it unsatisfactory for any but the mildest cases and in these cases protamine zinc insulin is much preferred because of its overlapping action.

Protamine zinc insulin is the best insulin in mild and moderately severe diabetics and gives best regulation in the patient whose fasting blood sugar before breakfast is high and the post-cibal "peak" blood sugar is not high. Vice versa of these blood sugars would be the very rare indication for globin insulin.

If the Protamine Zinc Insulin must be so greatly increased to control the blood sugar level during the day that it excessively reduces the early A. M. sugar level and causes hypoglycemic shock then, or globin insulin must be given in such large doses to control the early A. M. hyperglycemia that it causes late afternoon hypoglycemic shock, then we must turn to the so-called mixed insulin given as a single dose before breakfast. This is a mixture prepared from regular insulin mixed with protamine zinc insulin always of the same concentration in units, in ratio of about 2 to 1. This insulin has an intermediate action (as you can see from table 1) between globin and protamine zinc insulin. We do not use this mixture in the old person or young child (unless the child is of the bookworm type and does not exercise much). It is the choice of insulin for moderate to severe diabetics between the ages of 18 years and 50 years, and may be used after the age of 50 years, if there is no evidence of cardiovascular disease[12, 15], and it should be borne in mind that any poorly controlled diabetic of 10 years or more duration usually already has cardiovascular disease even though they have no symptoms of such.

In this changeover from regular or protamine zinc insulin to 2:1 mixed insulin, the total dose in units is reduced one third at start of the replacement; then if daytime glycosuria and hyperglycemia persist, the regular insulin content of the mixture is increased: e. g., making the ratio 2½:1 or 3:1. If the postabsorptive or fasting glycosemia and hyperglycemia persist after this changeover, then increase the protamine zinc insulin content of this mixture, e. g., making ratio 1½:1 or 1:1. Thus it is seen that the criteria for the adjustment of each component is identical with those criteria governing each component used alone.

An alternative method of starting the use of 2:1 mixed insulin, and one that will probably be used most in office or outpatient department patients, after starting the diet and four urinalyses daily, is to put them on a total of 9 to 21 units of a 2:1 mixture given before breakfast, and then revise this up or down depending on the response.

In the use of mixed insulin, the carbohydrate of the diet should be divided equally among the three meals. The great advan-

tage of mixed insulins is the avoidance of wide swings in the blood sugar level and control of the glycosuria.

When acute complications, such as an acute febrile illness, develop in a diabetic it is best to divide the diet and insulin (using only regular or crystalline insulin) into four or six equal amounts regularly spaced in the twenty-four hours of the day. Urine should be tested every four hours and insulin adjusted and given before each feeding; for example, in mild diabetics give 1500 or more calories, with plenty of fluids divided into six feedings, with six units given before each feeding, and decrease or increase this amount of regular insulin according to the four hour urine reactions.

Severe diabetic acidosis is best treated immediately and intensively in the hospital as it requires coordinated medical, nursing and laboratory efforts. Hypoglycemic reactions are likely to occur in any patient treated with insulin. They may be severe and even fatal. They occur: (1) Late at night or early in A. M. or during heavy exercise when protamine zinc insulin is used. (2) Late in P. M. if globin is used. (3) One or two hours after regular or crystalline insulin is given. This may occur in any patient taking insulin, and the diagnosis must be considered if there is a blood sugar below 70 mgm. per cent, though symptoms of shock often do not appear until the blood sugar is 40 mgm. per cent or below. The symptoms though variable follow a reproducible pattern in each individual. A blood sugar of 50-70 mgm. per cent gives nervousness, sweating, excitation, tremor, hunger, and, if uncontrolled, convulsions and coma; if 40 mgm. per cent or below weakness, headache, nausea, depression and convulsions.

It appears that the brain is involved from the cerebral cortex downwards and that there is a reduction of oxygen consumption hence from 6.8 vol. per cent to 2.57 to 1.77 and this is probably the cause of the symptom complex.[4] The following is a step by step outline of symptoms and signs of each progressive step in involvement of the brain cerebral cortex and cerebellum: Somnolence, sweating, salivation, tremors, excitement and confusion. Then thalamus: Sympathetic nervous stimulation, dilated pupils, tachycardia, flushing. The mid-brain: Reaction to stimulant resulting in tonic spasm and a positive Babinski sign, and finally involvement of the medulla oblongata. Increased parasympathetic activity: Pin point pupils—nonreactive to light, slow heartbeat, slowing of respiration, relaxation of muscles and the loss of corneal reflex. Of course, use glucose before this develops, but if this state is present also give barbiturate to control muscular contractions.

Pregnancy complicating diabetes is influenced most by an imbalance of sex hormones as Priscilla White of Boston has pointed out most conclusively. In the pregnant diabetic, high fetal mortality, pre-eclampsia and obstetrical abnormalities parallel an abnormal pattern of sex hormones. What should be a practical and up-to-date management of pregnancy in diabetes mellitus? First, pregnancy should be advised against in the diabetic over thirty years of age, or in diabetics of twenty-five years duration, as maternal morbidity is certain and maternal survival a gamble.

If already pregnant, or we allow pregnancy, a strict regime including chemical management is carried out. This includes restriction of salt and sodium bicarbonate intake, a high protein diet, 60-120 grains of ammonium chloride daily if the patient has edema, and substitutional estrogen and progesterone therapy. If a diabetic pregnant patient presents a history of an obstetrical accident, or upon appraisal appears to have hypogonadal stigmata, such as periods of amenorrhea, menorrhagia, chronic cystic mastitis, edema, or if weekly hormone analyses show a fall in serum level of estrogen, lowered excretion of pregnandiol glycuronate and compensatory rise in serum chorionic gonadotropin, hormone therapy

is indicated. The dosage of stilbesterol and progestoral in such cases, is as follows:

Up to 20th week: 5 mgm. daily
20-24th week: 10 mgm. daily
24-28th week: 15 mgm. daily
28-32nd week: 20 mgm. daily
32-36th week: 25 mgm. daily
36-delivery: 30 mgm. daily.

Premature delivery at the end of the thirty-seventh week is advocated; two thirds will require Cesarian section, under spinal anesthesia without preliminary sedation. If normal delivery occurs, medication should be kept to a minimum. Special care is required for the infant and to guard against atelectasis, and includes use of oxygen and insurance of its delivery to the baby's lungs.

Priscilla White in Boston reports three groups of cases that are convincing for the results obtained by hormone therapy.

TABLE 4

Diabetic Pregnancies in which	No. Cases	Maternal survival	Fetal survival	Pre-edompsia	Premature Deliveries
Hormone balance normal	66	100%	97%	2%	0
Hormone balance abnormal	58	100%	47%	50%	40%
Hormone balance abnormal but estrogen and progesterone supplied	174	99.5%*	90%	5%	5%

* One maternal death in this group due to infectious hepatitis.

Simple matters as proper care of feet, hands, and skin often prevent gangrene in A S diabetics. They should be instructed in these, by their physician. Exercise in moderation should be allowed, as it increases carbohydrate tolerance. Over fatigue, both mental and physical, and worry should be avoided, as they decrease carbohydrate tolerance and predispose to infection.

We have presented, as we see them, some of the more recent advances in pathological physiology, and in the more exact treatment of diabetes mellitus. We hope it will spur you on to a more aggressive attack on this problem, especially in your middle-aged and pregnant diabetics.

REFERENCES

1. Joslin, E. P.: Diabetes mellitus. New England J. Med., 236:474, 1947.
2. Ricketts, H. T.: Does hyperglycemia harm diabetic patient? Med. Clin. N. A., Mar. 1947.
3. Gordon, D. L.: The pathological physiology of diabetes mellitus. New Orleans M. and S. J., 99:451, 1947.
4. Weinstein, Albert: Diabetes mellitus. J. Tennessee
5. Marks, Herbert H.: Recent statistics on diabetes and diabetics. Med. Clin. N. A., Mar. 1947, p. 385.
6. Joslin et al: Treatment of diabetes mellitus. 1946. Lea and Febiger.
7. Rusk, H. A.: Rapid advance in research to aid diabetics is traced. New York Times, p. 30, Feb. 1, 1948.
8. Loef, Robert F.: Diabetes mellitus. Cecil's Textbook of Medicine: 7th Ed. 1947 Saunders, pp. 685-710.
9. Rickets, H. T., Brunschwig, A., and Knowlton, K.: Effects of total pancreatectomy in a patient with diabetes. Am. J. of Med. 1:229, Sept. 1946.
10. Combined Staff Clinics: Hypoglycemia. Am. J. of Med., 1:412, Oct. 1946.
11. Feldman, F., Roberts, J. B., Susselman, S., and State M. A., 40:220, 1947.
Lipets, B.: Coincidence of diabetes mellitus and hypopituitarism. Arch. Int. Med., 79:332, 1947.
12. Sterns, S., Schlesinger, M. J., Rudy, A.: Incidence and clinical significance of coronary artery disease, in diabetes mellitus, Arch. Int. Med. 80:463, 1947.
13. Henderson, L. L., Sprane, R. G., and Wingener, H. P.: Intracapillary glomerulosclerosis. Am. J. Med. 111:131, 1945.
14. Shapiro, W. W., and Fenoke, H. O.: Medical control of diabetes in the surgical patient, American Practitioner 269, 1947.
15. Scoville and others of Vanderbilt University Hospital Medical Staff, Nashville, Tenn. Personal communication.
16. Peck: Med. Clin. of N. A., March 1947.
17. Franks, M., Berris, R. F., Koplan, N. O., and Myers, G. B.: Metabolic studies in diabetic acidosis, Arch. Int. Med. 80:739, 1947.
18. Franks et al ibid, 81:42-56, 1948.
19. White, P.: Pregnancy complication diabetes, J. A. M. A. 128:181, 1945.
20. White, P.: Pregnancy complicating diabetes, Pennsylvania M. J. 50:705, 1947.
21. Mayerson, H. S.: Head of Dept. of Physiology, Tulane Medical School, New Orleans, La. Personal communication.
22. Joslin, et al: Medical Progress: Diabetes. New England J. Med. 238:401, 1948.
23. Ibid 238: Mar. 25, 1948, No. 13.
24. Ibid 238: Apr. 1, 1948, No. 14.

AN EVALUATION OF THE USE OF VARIOUS INSULIN PREPARATIONS IN DIABETES MELLITUS

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The discovery of insulin marked a tremendous advance in the treatment of diabetes mellitus. Amorphous insulin is not

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entirely satistactory in the control of human diabetes mellitus. Various modifications of the original product have been used in an endeavor to convert the diabetic into a more normal organism.

Because of the difficulties encountered in the use of insulin and its various modifications, two schools of treatment have arisen. The one, headed by Tolstoi[1, 2], relaxes the standards of control considerably in severe cases and philosophically tolerates pronounced glycosuria provided ketosis, loss of weight, increased thirst and polyuria are absent. It is Tolstoi's contention that the amount of carbohydrate utilized is vastly more important than the amount of sugar excreted in the urine. The other group attempts to adapt the patient to his insulin by altering the distribution of the carbohydrate and protein in meals, by supplementary feedings through the 24 hour period, by the administration of one or more types of insulin in several injections daily, etc. Dietary juggling is tedious; it necessitates a reasonable familiarity with dietary principles, complicates the usual hospital routine and may require alterations of the food habits of patients. Multiple feedings and injections are hardly conducive to a "normal" existence.

The question to be answered is: Is it necessary, or even desirable, to avoid hyperglycemia and glycosuria? While it is not universally accepted that lax control of the disease is responsible for the degenerative complications which characterize it, there is suggestive evidence[3] both in animals and man that this may be the case. In the absence of proof to the contrary, it may be hazardous to abandon the goal of normoglycemia in the treatment of diabetic patients.

The search for an "ideal" insulin preparation continues. Its requirements have been outlined by Colwell,[4] "On purely theoretical grounds, the ideal insulin is one whose solubility and rate of insulin release is such that it acts promptly and intensely enough to control the glycosuria following meals, but does not cause hypoglycemia between meals. It must act long enough to control nocturnal hyperglycemia but also wane rapidly enough to avoid nocturnal hypoglycemia. It should possess the greatest possible constancy of action from dose to dose without sacrificing prolongation of effect." Although such an insulin preparation has not been produced as yet, encouraging progress has been made toward this goal.

The purpose of this paper is to present the characteristics of the various insulins at our disposal and the results of treatment in order to evaluate their advantages, as well as their shortcomings, in diabetic therapy. This should serve to facilitate and simplify the management of these patients.

Four types of insulin have become available for the routine treatment of diabetes mellitus. These are: (1) unmodified insulin, (2) protamine zinc insulin, (3) globin insulin with zinc, and (4) mixtures of regular and protamine zinc insulin and modifications of protamine zinc insulin.

1. UNMODIFIED INSULIN

This is the true hormone as discovered by Best and Banting in 1921, and crystallized by Abel in 1925. All other insulins in use today stem from this substance which has been altered in one way or another to produce prolongation of effect although the essentials of its action on carbohydrate metabolism are unaltered.

Insulin is an amphoteric substance. It has an isoelectric point at which it exhibits minimum solubility. (pH6.0-6.2). Beyond that point it is readily soluble. A crystalline insulin has been produced whose solubility is complete only at a pH greater than 6.4. Its proposed advantages[5-7] may be stated in these simple terms: An insulin of greater alkalinity is absorbed more slowly by the tissues and the blood, and this in turn slows down the glycolytic effect of the insulin molecule. That the slight prolongation of effect is not entirely attributable to increased zinc content has been shown by Altschuler and Leiser.[8]

Marble and Vartiainen[9], in a series of experiments, obtained blood sugar curves which showed the effect of zinc insulin crystals to be almost identical with amor-

phous (regular) insulin. In fasting diabetic subjects, the action of crystalline insulin was slightly prolonged, producing a slightly longer blood sugar lowering effect than can be obtained with amorphous insulin. The lowest point in the curve following a single injection is reached in 6 hours with amorphous (regular) and in 7 hours with crystalline insulin. Duration of action was 9.6 hours for the amorphous and 11.2 hours for the crystalline insulin. On maintenance doses, however, the differences were of such slight degree as to be of minor clinical significance. In such cases there was demonstrable effect within 1 hour with peak action between the third and sixth hour and an effect which was demonstrable for 8 to 12 hours.

Advantages: Because of its solubility, unmodified insulin acts most briefly but is the most prompt and intense of the insulins and exhibits greatest uniformity of effect. For these reasons, unmodified insulin is the preparation of choice in emergencies where these qualities of action are mandatory, e.g. in acidosis, infections, following trauma, and preoperatively and postoperatively. It is useful in supplementing protamine insulin effects when indicated. It is also indicated in those cases in which control is impossible with one of the types of protamine insulin.

Disadvantages: These include short duration of action which necessitates injections every four to six hours throughout each twenty-four hour period. However, as emphasized by Joslin,[11] this is not its only drawback. As a matter of fact, at least for half of its effective period, it is overactive and underactive because a preliminary time is required for it to exert an adequate effect and later hypoglycemia can easily result. In other words, there are alternating periods of hyperglycemia and hypoglycemia throughout each twenty-four hour period.

An occasional disadvantage of amorphous (regular) insulin, is the occurrence of allergic reactions associated with contaminating proteins. These reactions may be avoided by the use of the purer crystalline insulin.

2. PROTAMINE INSULIN

Hagedorn and his co-workers[12, 13] at the Steno Memorial Clinic in Copenhagen conceived the idea that if insulin could be combined with some basic group so that the pH of the combination would be nearer the pH of tissue fluid, then absorption might be slowed. In this compound, insulin acts as an acid, whereas in insulin hydrochloride it acts as a base. For his purpose, he resorted to the protamines, and the most suitable of these proved to be that derived from the sperm of rainbow trout. This combination alone was insufficient to delay absorption but when the reaction of the solution was adjusted to a pH of 7.2, which is similar to that of tissue fluid, a precipitation of protamine insulin took place. As the result of the compound being a suspension, the deposit in the subcutaneous tissue consisted of (1) a fluid of practically constant insulin concentration from which absorption takes place and (2) a steadily diminishing amount of solid particles. In other words, the compound was slowly broken down and active insulin released over a relatively long period of time thus allowing for a more even and prolonged effect on the blood sugar.

Clinical Use: This insulin was subjected to extensive clinical trial by numerous competent observers[14-19] who confirmed the results of Hagedorn. The duration of action of a given dose is approximately fourteen hours. Thus a single daily dose is sufficient for adequate control in many cases. In those diabetics not satisfactorily controlled by a single daily injection, Hagedorn[12, 13] found that the administration of regular insulin each morning before breakfast and the injection of protamine insulin before retiring insured much more adequate control. The purpose of this regime was to prevent hyperglycemia and glycosuria from occurring in the forenoon because of the slower action of the newer insulin and, at the same time, to satisfactorily control nocturnal hyperglycemia. Similar results were

obtained by Joslin and his group,[14, 15] Lawrence and others.[16]

Certain dietary adjustments were necessary to obtain the most satisfactory results with this insulin. As stressed by Campbell,[17] diets very high in carbohydrate and low in fat are less suitable than diets high in fat and low in carbohydrate. Joslin[14, 15] divides the total carbohydrate of the diet into two-fifths for breakfast and lunch and one-fifth for supper, in order to more readily control the blood sugar after the evening meal.

Advantages: These include (1) prolonged action which insures better control with fewer injections and (2) reduced liability to hypoglycemia because of its slower and less violent action. "Whereas the Banting era made possible the conquest of coma, the Hagedorn era makes possible the physiological processes of the diabetic to be more nearly normal." (Joslin[15]).

Disadvantages: These include (1) instability of the protamine compound if kept for more than three or four weeks; (2) because the preparation is a suspension, thorough mixing is essential in order to assure identical dose measurements; (3) its *inability* to adequately control the marked hyperglycemia and glycosuria following high carbohydrate meals. In other words, rapidity of onset, intensity of effect, and constancy are sacrificed for duration of effect as compared with unmodified insulin; (4) dietary rearrangement with its various disadvantages is essential for best results; (5) because of its peculiarities of action, this insulin is not advisable in situations in which immediate and intense insulin effect is desired.

3. PROTAMINE ZINC INSULIN

The discoverer of this insulin is Scott[20] of Toronto, who showed that crystalline insulin is a true chemical compound of zinc and insulin-zinc insulinate. When he turned his attention to protamine insulin compounds he found that when entirely freed from zinc and traces of allied metals, they were more prolonged in their action than soluble insulin hydrochloride. The addition of traces of zinc and adjustment of the pH to 7.2 produced a zinc protamine-insulin compound in suspension which was more stable and more prolonged in its action than any previously discovered. Extensive clinical study by Rabinowitch and his group[21] served to support Scott's claims. As suggested by Bang,[22] zinc delayed and prolonged the rate of insulin release beyond that observed by Hagedorn, possibly by inhibiting an enzymatic process responsible for the separation of insulin from its precipitating protein in tissue fluid.

Chemistry: Protamine zinc insulin is a cloudy suspension which contains 0.75 to 1.25 mgm. of protamine and 0.2 mgm. of zinc per 100 units of insulin buffered to a pH of 7.2. Standard protamine zinc insulin contains an excess of 40 per cent protamine. This is because precipitation is not complete. In order to precipitate practically all of the small amount of insulin remaining in solution and to place the solubility of the combination well beyond the point where minor variations may result in significant alteration of the rate of activity, it was decided to follow Hagedorn's advice and include excess protamine. This excess also has the effect of combining with the tissue proteins about the depot of injection thereby delaying absorption still further and increasing the prolonged action of the product (Peck[23]).

Time Activity: Careful studies of the time activity of protamine zinc insulin have been carried out by Eastman and Greeley[24] and Martin and Greeley.[25] Time activity curves obtained following the administration of 50 units of protamine zinc insulin to a depancreatized dog revealed the following: The onset of activity is slow being demonstrable in three to four hours; a delayed peak of activity occurred between seventeen and twenty-four hours following the administration of insulin, and the duration of action is approximately seventy-two hours. This curve is similar in general to a curve following the administration of 100 units of protamine zinc insulin to a severe, fasting diabetic.

As regards the duration of action and activity of different size doses of protamine

zinc insulin, several conclusions were reached by these observers: (1) There is a difference in duration of action of a given dose of protamine zinc insulin—in the mild diabetic with no basal insulin requirement the duration is longer than in the severe diabetic with a basal insulin requirement. (2) There is a period of maximum activity with all size doses of protamine zinc insulin and this period is delayed with the larger doses; and (3) it is important to give protamine zinc insulin to the severe diabetic properly timed in relation to the period when maximum insulin effect is desired.

The latter observations have been challenged by Ricketts[26] in an interesting study. Eight severe, well controlled diabetics were given their calculated diets daily and prescribed dosage of protamine zinc insulin daily at 7:30 A. M. Composite curves were obtained by examination of the blood sugar at regular intervals. After a suitable rest period, protamine zinc insulin was given at 8:00 P. M. and composite blood sugar curves were obtained. In 3 patients studied, the administration of protamine zinc insulin in the evening resulted in blood sugar curves practically identical with those observed during the morning period. In 4 of the remaining 5 patients, the differences between the morning and evening curves were slight, and since they did not lie consistently in one direction are probably to be explained on the basis of variations in the state of the diabetes from one week to another rather than by the superiority of one regime over another. This constancy of action of protamine zinc insulin holds several practical and theoretical implications: (1) It means that this form of insulin should not be regarded as having a period of maximum effectiveness. While it lowers the blood sugar to a minimum in twelve to eighteen hours, when given as a single dose to patients not receiving food, it furnishes a continuous and relatively even supply of insulin from the subcutaneous depots when used in the day-by-day treatment of diabetes. Its action, once fully established, is maximal at all times. The patient, therefore, can take his injections at whatever time of day it is most convenient, provided that they are twenty-four hours apart. (2) If nocturnal hypoglycemia occurs, it is not because of any greater insulin action at this time but because the amount is so large as to render the basic level too low. (3) It controls the mild case of diabetes admirably but in the severe case, even though it may render and maintain the blood sugar normal during fasting, it does not completely prevent hyperglycemia after a mixed meal. This would suggest that the chief function of protamine zinc insulin is in the regulation of endogenous carbohydrate metabolism.

Since a given dose of protamine zinc insulin exerts some effect for as long as seventy-two hours, changes in the dose administered should not be any more frequent than every third or fourth day.

Use: Protamine zinc insulin has been subjected to widespread clinical trial and critical analysis by numerous competent observers.[1, 2, 27, 47] It has become increasingly obvious that this insulin has become invaluable as a means of providing sustained insulin effect with one daily dose. Mild diabetics, i.e. those who require 40 units or less of insulin daily, are readily controlled, whereas a larger percentage of severe diabetics are not well controlled with a single daily injection of protamine zinc insulin. Even in severe diabetes, however, control is vastly superior to that obtained with multiple daily injections of regular insulin. Response to this type of insulin therapy is much more gratifying if the diet contains no more than 150 Grams of carbohydrate daily. In order to assure maximum efficiency with protamine zinc insulin, the total carbohydrate of the diet is best divided into one-fifth, two-fifths and two-fifths, for breakfast, lunch, and supper, respectively, with a glass of milk and a slice of bread, or one or two crackers, reserved for a bedtime snack.

Advantages: These include: (1) Slow and persistent insulin action over a twenty-four hour period making it possible for a mild diabetic to metabolize his carbohy-

drate in a normal fashion throughout this period. (2) The fact that the action of protamine zinc insulin is exerted over a period longer than twenty-four hours enables the patient to begin his day with a normal blood sugar. This is what Joslin terms as "insulin insurance from one day to the next." (3) Because of its characteristics of action, diabetic coma is inexcusable in all but a small fraction of patients. (4) The compound is quite stable.

Disadvantages: (1) Protamine zinc insulin provides a constant small amount of circulating insulin sufficient to control endogenous carbohydrate metabolism but too weak to control the blood sugar after high carbohydrate meals. Increasing the dose of protamine zinc insulin to control postprandial hyperglycemia will result in unpleasant, and often dangerous, nocturnal hypoglycemia. (2) Dietary readjustment, with its various trials and tribulations, is essential for best results. (3) Hypoglycemic reactions with protamine zinc insulin are mild and gradual in onset and are definitely more difficult to detect and treat. The fact that these reactions are more prone to occur after the patient has retired adds to the hazard. Insulin reactions with protamine zinc insulin occur at much lower blood sugar levels than with regular insulin. This is possibly because the drop in blood sugar level is so gradual that the adrenals and sympathetic nervous system are not stimulated to secrete adrenalin and promote mobilization of liver glycogen. It should also be borne in mind that whereas tremor and perspiration are the most common symptoms of a "reaction" from regular insulin, patients receiving protamine zinc insulin most often complain of headache, usually occipital, nausea, and vomiting. These symptoms may persist for hours and interfere greatly with the intake of food. These are often symptoms of acidosis, and care must be exercised in differentiating between the two states. (4) The sudden onset of any condition preventing the intake of food following the daily injection of protamine zinc insulin is another disadvantage of its slow and prolonged action. (5) This is not the insulin to be used in diabetic emergencies where rapid and intense insulin effect is desired. (6) Local allergic reactions are not infrequent and are, at times, annoying. (7) A certain minor disadvantage is the fact that since protamine zinc insulin is a suspension, the vial must be carefully and gently shaken before the withdrawal of each dose to insure a uniform dose.

As in the case of protamine insulin, protamine *zinc* insulin has sacrificed rapidity and intensity of action and constancy of effect for prolongation of activity. Obviously, a certain number of cases will be poorly controlled on protamine zinc insulin.

4. GLOBIN INSULIN WITH ZINC

Since the introduction of slowly acting protamine zinc insulin in 1937, many attempts have been made to modify and improve its action. These investigations have been stimulated primarily by the recognition that one-third to one-half of the patients using protamine zinc insulin require a supplementary injection of unmodified insulin to insure a satisfactory blood sugar during the earliest part of the day. This has been necessitated by the lack of "rapid, early effect" of protamine zinc insulin.

The first attempt to improve on protamine zinc insulin without sacrificing its obvious advantage of sustained action was the introduction in 1939 of globin insulin. The preparation was developed by Reiner, Searle and Lang[48] and studied clinically by Bauman,[49-51] Andrews and Grant,[52] Duncan and Barnes,[53] Bailey and Marble[54] and others.[55-62]

Chemistry: According to Reiner, Searle, and Lang,[48] when insulin was mixed with globin derived from beef hemoglobin, the solution remained clear at a pH of 4 or less and precipitated between pH 5 and 8. When 0.2-0.3 mgm. of zinc was added to a preparation containing 3.8 mgm. of globin per 100 units of insulin, the duration of hypoglycemia was more than twice that obtained with amorphous insulin containing the same amount of zinc.

At the present time, globin insulin with

zinc as manufactured commercially is a clear, colorless solution with a pH of 3.7 and composed of 3.04 mgm. of globin and 0.24 mgm. of zinc per 100 units of insulin.

Action: Careful studies of the time activity of globin with zinc were carried out by Martin, Simonsen, and Homann.[58] These revealed that the onset of action of globin insulin occurred within one to two hours after injection with its peak occurring between the fifth and tenth hours. Action is apparent for eighteen to twenty hours although the action of globin insulin is not marked beyond the fourteenth hour. However, as stated previously, the duration of action is a function of the size of the dose and the severity of the diabetes.

There are important clinical applications to be drawn from these studies. First, patients are much better controlled if the total daily carbohydrate is divided in such a manner that the greatest intake coincides with the greatest intensity of insulin action. For this reason, the total carbohydrate is usually divided one-fifth, two-fifths, and two-fifths, for breakfast, lunch, and supper, respectively. Second, the most intense action occurs five to ten hours after injection and hypoglycemic reactions are most likely to occur in the early or middle afternoon, i.e., between 2:00 P. M. and 4:00 P. M. For this reason, a small afternoon snack, such as a glass of milk and one or two crackers or a slice of bread, is necessary in order to prevent the occurrence of any hypoglycemic reaction. Best results are obtained if the total daily carbohydrate does not exceed 150 Grams.

It is now acknowledged by the majority of clinicians that the mild diabetics, i.e., those requiring 40 units or less of insulin daily, are well controlled by a single daily injection of globin zinc insulin. The severe diabetic, however, is still a problem and, whereas many of these cases will respond better to globin than to protamine zinc insulin, they are still poorly controlled and will require supplementary injections of unmodified insulin for best control. Andrews *et al*,[52] however, feel that globin is the insulin of choice, particularly in severe diabetics.

Advantages: (1) Globin insulin is particularly valuable in regulating patients who have a rise in blood sugar after eating only, and not during the night after fasting. (2) Because of its peculiarities of action, hypoglycemic reactions, when they occur, are at a time when they can be promptly treated. A sense of weakness and chilliness was the outstanding symptom of hypoglycemia in many patients, the latter often persisting for an hour or more after all other symptoms had disappeared. (3) Fewer allergic reactions occur with globin insulin which causes it to become particularly valuable to those diabetics who are allergic to protamine zinc insulin. (4) It is a clear solution. (5) It is valuable as a supplement to protamine zinc insulin in the control of those patients whose fasting blood sugar is normalized by the latter but who require an additional daytime adjuvant to prevent postprandial hyperglycemia and glycosuria.[57] (6) It controls practically all mild diabetics with one daily injection.

Disadvantages: (1) Its supposed advantage of homogenicity in solution appears undesirable since the acid solution is precipitated by the alkaline tissue fluid on injection with resulting inconstancy of action from dose to dose, depending on completeness of precipitation in individual depots (Colwell). (2) As stated by Peck, "Acid, or clear, insulin modifications seem more likely to dump out their insulin at odd moments and result in unexpected hypoglycemic reactions." (3) Daily morning injections fail to permit the overlapping effect so essential for good control. (4) Its hourly carbohydrate-handling ability is too low to cover the diet in most severe diabetics. (5) Many patients complain of marked burning on injection. (6) Dietary juggling with its various disadvantages, is essential for best results.

It should be noted that in more rapid and intense action than that obtained with protamine zinc insulin, duration of effect has been sacrificed. In addition, action is not intense enough to control exogenous

carbohydrate metabolism in the severe diabetic and is sufficient in the mild diabetic only through certain dietary adjustments.

5. INSULIN MIXTURES AND MODIFICATIONS

Much work on mixture of soluble and protamine zinc insulins and various modifications of protamine zinc insulin has been carried out by MacBryde and Roberts[63, 64], Colwell[4, 65-67], Peck[23, 68-73]; Ulrich[74], and others[75-81]. Lawrence[40, 41] was probably the first to suggest the mixing of regular insulin and protamine zinc insulin in the same syringe. As early as 1937, Lawrence wrote: "A combination of the two types of insulin action is needed to turn the severe diabetic into a physiological organism and this has not yet been achieved by any single preparation. The next best thing, however, is to mix soluble and suspension insulin in correct proportions and give them in one injection. So far, a mixture of these two insulins in one bottle has not been produced, but we have satisfied ourselves by clinical experiments that soluble and suspension insulin can be given in the same syringe and injected into the same place and each retains its characteristic action." The "correct proportions" consist of that amount of protamine zinc insulin which is necessary to control endogenous carbohydrate metabolism to which is added that amount of soluble insulin sufficient to control postprandial hyperglycemia. Those cases cited had either a slight excess of protamine zinc insulin or equal parts of protamine zinc and soluble insulin.

Ullrich,[74] in 1941, in a series of experiments, showed that with mixtures of equal parts of protamine zinc insulin and regular insulin there was an effect definitely different from that which would be obtained from corresponding doses of protamine zinc insulin and regular insulin injected separately. For example, since there is approximately 40 per cent excess protamine in protamine zinc insulin, mixing 1 cc. u-20 regular insulin and 1 cc. u-20 protamine zinc insulin would produce a resultant mixture containing 28 units of protamine zinc insulin and 12 units of regular (uncombined) insulin provided protamine combines quantitatively with added free insulin. The action obtained from this mixture is less immediate than that obtained with separate injections of protamine zinc insulin, 28 units, and regular insulin, 12 units, but was definitely greater than with protamine zinc insulin alone.

The combination of protamine zinc insulin and regular insulin in mixture can hardly result in 1:1 combination since Colwell[66] and his associates showed that even when insulin is added in excess over protamine zinc insulin up to 4:1 proportions, little of it remains in the soluble form in the supernatant liquid. In other words, even when as much as 64 units of soluble insulin is mixed with 16 units of commercial protamine zinc insulin, only about 3 units, or one-twentieth of the added insulin, stays in solution. Thus the accelerated and more intense action of such mixtures cannot be due to ordinary insulin in solution.

The explanation of this action, which is probably most correct, conceives of an entire series of protamine zinc *insulins*, of which the commercial product is only one, in which increasing amounts of insulin may be combined in an increasingly "insulin-saturated" insoluble complex up to the saturation point. This series of precipitates has the following properties: (1) more prompt and intense and less prolonged activity than commercial protamine zinc insulin; (2) monophasic action suggesting insulin in a single complex rather than in two compounds; (3) little insulin in solution even when added in 4:1 excess or in 2:1 excess when buffered to a pH of 7.2; and (4) excess insulin is combined firmly in a protamine zinc insulin complex because it does not dissolve on buffering to a pH of 7.2 unless buffered immediately after solution. This would nullify Ullrich's explanation[74] that added insulin may not be combined firmly with protamine accounting for difference in action.

The preparation obtained by mixing protamine zinc insulin with regular insulin has a pH between the two. Both Colwell[4, 65-67] and Peck[23, 68-73] believe that these mixtures are stable for many months and that their

action does not vary appreciably when they are freshly mixed in the syringe on injection. Palmer[81] believes that it is better to prepare a mixture in one vial than to prepare it daily in one syringe whereas Colwell, Peck, and the great majority of other workers in this field prefer to mix the insulins in one syringe daily.

MacBryde and Roberts[63, 64] approached the problem somewhat differently working with a specially prepared insulin containing 0.5-0.625 mgm. of protamine and 0.1 mgm. of zinc per 100 units of insulin and buffered to a pH of 7.2. This turbid suspension contained approximately 25 per cent rapidly acting (soluble) insulin and 75 per cent slowly acting (precipitated) insulin and is commonly referred to as "NP50". Recently, the stability of the soluble insulin in this preparation has been questioned which, if true, might make the results with this insulin variable depending on its age since fresh lots of insulin would contain a larger amount of soluble insulin than the older ones. MacBryde is continuing his effort to improve this insulin.

Time Activity: In effect, a mixture containing equal portions of the two insulins is barely distinguishable from protamine zinc insulin; a mixture containing four parts of soluble insulin and one part of protamine zinc insulin resembles ordinary insulin. Mixtures of 2:1 and 3:1 show intermediate effects—there is demonstrable activity within four hours, peak effects in eight to sixteen hours, and definite waning of effects after twenty-four hours. The intensity of action at its peak is intermediate between regular and protamine zinc insulin.[65, 68, 76]

The percentage of action expended in the first twenty-four hours for the various insulins according to Peck[71], is as follows: (1) protamine zinc insulin 34 per cent; (2) 2:1 (Regular PZI) mixture 58 per cent; (3) 3:1 mixture 60 per cent; (4) 4:1 mixture 64 per cent; (5) globin insulin with zinc 74 per cent; and (6) crystalline or regular insulin 80 per cent.

Use: Those mixtures most commonly used are 2:1 and 3:1. Peck and Schecter[71] in 1944 reported 150 cases using extemporaneous mixtures with the following results: 6 per cent were satisfactorily managed with a 1:1 combination, 16 per cent with a 3:2 mixture, 70 per cent with a 2:1 mixture, and 8 per cent with a 3:1 mixture. Subsequent experience[72] has shown a further decrease in the number of cases requiring less than a 2:1 mixture so that the proportion of cases now taking 2:1 combinations has enlarged still further.

Colwell's[67] most recent experience in a group of 150 severe diabetics revealed 85 per cent improvement on 2:1 and 3:1 mixtures. Hildebrand and Rynearson,[77] Palmer[81], and Sparks and John[76] found that the 2:1 mixture was most commonly and effectively used, whereas Ullrich[74] prefers a 3:2 mixture. Of interest is a series of 80 cases reported by Aldersberg and Dolger[80] in which 35 per cent of their cases were well regulated on "surplus protamine" mixtures (ratios 3:4, 2:3, 1:3, etc.) whereas only 21 per cent were regulated on "surplus regular" mixtures (ratios 2:1, 3:1, 3:2, etc.)

MacBryde and Roberts employing "NP50" reported 90 per cent success in 110 cases and del Fierro and Sevringhaus[79] reported excellent results in 14 of 16 cases. As stated by Peck,[72] the degree of clinical improvement with a 2:1 mixture, as advocated by Colwell, and the specially modified protamine zinc insulin, as advocated by MacBryde and Roberts, was for practical purposes equal. The general character of the curve obtained with the 2:1 mixture was smoother and subjected to less periodic variation but both curves were well within normal limits. As stated previously, however, NP50 has not been developed to such an extent as to meet rigid market reuirements.

The method of regulation as described by Peck is as follows: Replace multiple injections with single dose admixture: (a) Replace total with 2:1 mixture; (b) If daytime glycosuria and hyperglycemia (2 hours pc) persists, increase insulin content, e. g. 2½:1, 3:1, etc.; (c) If fasting glycosuria and hyperglycemia persists, increase amount of PZI in mixture, e. g., 3:2.

The technic of mixing the two insulins is easy to do but difficult to explain. Patients must actually be shown how to make their mixtures. The new feature to be learned is how to draw an air bubble into the syringe and roll it through to mix the doses.[73] It must be emphasized that the dose of unmodified insulin is drawn into the syringe first. Since all the daily doses are combined in one injection, a preparation containing 80 units per cc. is preferable in order to lessen the volume of fluid injected. Preparations of the same manufacturer should be used in order to keep conditions as constant as possible.

It has been our experience that at least half of the patients requiring a mixture are eventually well regulated on fewer total units of insulin daily than previously. For this reason, we feel that it is probably desirable for the 2:1 mixture to contain approximately three-fourths as many total units to prevent undesirable hypoglycemic reactions. As suggested by Colwell, it has been possible, in a large series of severe diabetics to shift directly from protamine zinc insulin to a 2:1 mixture, when needed, without a single indication for a preparation intermediate between the two.

Colwell has stated that best results are also obtained with a diet containing carbohydrate and fat in the ratio of 3:2 in Grams, and response is better if the total daily carbohydrate is in the vicinity of 150 Grams. It is also possible with these modified insulins to eat three equi-caloric feedings daily although we have not insisted that patients previously using protamine zinc insulin and accustomed to a bedtime snack give up this meal.

As regards the choice between adjustable mixtures and the use of a premixed preparation such as NP50, the former seems quite preferable since the action of the preparation can be "tailor made" to fit each person's needs.[73, 76]

Advantages: (1) The action of these mixtures is rapid and intense enough to control postprandial hyperglycemia and glycosuria without producing hypoglycemia between meals; (2) Action is long enough to control nocturnal blood sugar but not intense enough to produce distressing hypoglycemic reactions during this time; (3) Action persists beyond twenty-four hours which permits the overlapping effect so essential for good control. (4) Hypoglycemic reactions are infrequent but, because of the peculiarities of the mixture, if they do occur, they are most common within the first 12 hours when they are easier to recognize and treat. (5) The use of these preparations obviates tedious dietary readjustments. (6) Only one daily injection of insulin is required; (7) It does not increase the number of insulins commercially available which would, in turn, further complicate the treatment of this disorder.

Disadvantages: A minor disadvantage is the difficulty which the patient may encounter when attempting to mix the two insulins in a single syringe prior to injection. We have, however, been able to instruct patients of low intelligence adequately in the Out-Patient Department with excellent results.

It would thus seem that these mixtures of regular and protamine zinc insulin, which are "tailor made" to fit the individual, exhibit those properties essential for the ideal insulin to possess. The indications for extemporaneous mixtures are those cases which are not adequately regulated by one daily injection of globin or protamine zinc insulins and who require multiple injections of regular insulin for adequate control.

REFERENCES

1. Tolstoi, E. and Weber, F. C., Jr.: Protamine zinc insulin: a clinical study, report of a group of diabetic patients in whose cases glycosuria was disregarded for one year, Arch. Int. Med., 66:670, 1940.

2. Tolstoi, E.: Newer concepts in the treatment of diabetes mellitus with protamine zinc insulin, Am. J. Digest. Dis., 10:247, 1943.

3. Ricketts, H. T.: Does hyperglycemia harm the diabetic patient? M. Clin. North America, pp. 267-277, March 1947.

4. Colwell, A. R. and Izzo, J. L.: Protamine zinc insulin modified for accelerated action, J.A.M.A., 122: 1231, 1943.

5. Barach, J. H.: Crystalline insulin, Ann. Int. Med., 10:1335, 1937.

6. Altschuler, S. S.: The clinical use of crystaline insulin, Ann. Int. Med., 11:901, 1937.

7. Levin, C. M., Kleefield, E. A. and Luciano, F. A.: Experience with zinc insulin crystals, J. Lab. & Clin. Med., 24:1030, 1939.

8. Altschuler, S. S., and Leiser, R.: The effect of

zinc content upon the action of insulins, Am. J. Med. Sc., 195:234, 1938.

9. Marble, A., and Vartiainen, I.: Crystalline insulin, J.A.M.A., 113:1303, 1939.

10. Ricketts, H. T., and Wilder, R. M.: Solutions of amorphous insulin and solutions of zinc insulin crystals: clinical studies on the comparative speed and duration of effect, J.A.M.A., 113:1310, 1939.

11. Joslin, E. P.: The use of insulin in its various forms in the treatment of diabetes, Bull. New York Acad. Med., 18:200, 1942.

12. Hagedorn, H. C., Jensen, B. N., Krarup, N. B., and Wadstrup, I.: Protamine insulinate, J.A.M.A., 106:177, 1936.

13. Hagedorn, H. C.: Protamine insulinate, Proc. Roy. Soc. Med., 30:805, 1937.

14. Root, H. F., White, P., Marble, A., and Stolz, E. H.: Clinical experiences with protamine insulinate, J.A.M.A., 106:180, 1936.

15. Joslin, E. P., Root, H. F., Marble, A., White, P., Joslin, A. P., and Lynch G. W.: Protamine insulin, New England J. Med., 214:1079, 1936.

16. Lawrence, R. D., and Archer, N.: Some experiments with protamine insulinate, Brit. M. J., 1:747, 1936.

17. Campbell, W. R., Fletcher, A. A., and Kerr, R. B.: Protamine insulin in the treatment of diabetes mellitus, Am. J. M. Sc., 192:589, 1936.

18. Wilder, R. M.: The new insulin, Minnesota Med., 20:6, 1937.

19. Barach, J. A.: Experimental studies on the effect of insulin, protamine insulin, and crystalline insulin, Pennsylvania M. J., 40:349, 1936.

20. Scott, D. A., and Fisher, A. M.: Effect of zinc salts on the action of insulin, J. Phar. & Ther., 55:206, 1935.

21. Rabinowitch, I. M., Fowler, A. F., and Corcoran, A. C.: Clinical experiences with protamine zinc insulin and other mixtures of zinc and insulin in diabetes mellitus, Can. M. A. J., 35:239, 1936.

22. Bang, H. O.: Enzymatic breakdown on protamine insulin, Acta Pharmacol et Toxicol., 2:89, 1946.

23. Peck, F. B.: Action of insulin, Proc. Am. Diabetes As., 2:69, 1943.

24. Eastman, H. E., and Greeley, P. O.: Time activity curves of protamine zinc-insulin: clinical application and significance of such curves in the treatment of patients with severe diabetes, Arch. Int. Med., 67:194.

25. Martin, H. E., and Greeley, P. O.: The duration of action and the activity of different size doses of protamine zinc insulin, Am. J. M. Sc., 203:402, 1942.

26. Ricketts, H. T.: The constancy of action of protamine zinc insulin, Am. J. M. Sc., 201:51, 1941.

27. Myers, G. B., and Perkins, F. S.: Comparative effects of the new insulin preparations upon the blood sugar curves, J. Lab. & Clin. Med., 23:1, 1937.

28. Rudy, A.: Clinical experience with protamine insulin in various modifications, Endocrinology, 21:544, 1937.

29. Wilder, R. M., and Wilbur, D. L.: Diseases of metabolism and nutrition, Arch. Int. Med., 61:316, 1938.

30. Wilder, R. M.: Clinical investigations of insulin with prolonged activity, Ann. Int. Med., 11:13, 1937.

31. Joslin, E. P.: Protamine (zinc) insulin, J.A.M.A., 109:497, 1937.

32. Joslin, E. P.: Diabetes and protamine (zinc) insulin, Mil. Surgeon, 82:1, 1937.

33. Joslin, E. P.: Difficulties in the use of protamine zinc insulin, J.A.M.A., 110:90, 1938.

34. Joslin, E. P., Root, H. F., White, P., Marble, A., and Joslin, A. P.: Protamine zinc insulin M. Clin. North America, 22:711, 1938.

35. Shepardson, H. D., and Friedlander, R. D.: Clinical experience with long acting insulin in ambulatory diabetic patients, Ann. Int. Med., 12:830, 1938.

36. Neuhoff, F., and Rabinowitch, C.: Protamine zinc insulin: Clinical observations and comparative analysis of blood sugar curves obtained with the use of protamine zinc insulin and regular insulin, Arch. Int. Med., 62:447, 1938.

37. Mosenthal, H. O.: Protamine zinc insulin—clinical application, J.A.M.A., 110:87, 1938.

38. Mosenthal, H. O., and Mark, M. F.: The prolonged use of protamine zinc insulin, J.A.M.A., 113:17, 1939.

39. Kepler, E. F.: Clinical experience with protamine zinc insulin, J.A.M.A., 110:92, 1938.

40. Lawrence, R. D., and Archer, N.: Zinc-protamine-insulin: a clinical trial of the new preparation, Brit. M. J., 1:487-491, 1937.

41. Lawrence, R. D.: Zinc-protamine-insulin in diabetes: treatment by one daily injection, Brit. M. J., 1: 1077, 1939.

42. McCullogh, E. P.: Protamine zinc insulin in diabetes, Ann. Int. Med., 11:1979, 1938.

43. Rynearson, E. H.: Practical suggestions for the use of protamine zinc insulin, M. Clin. North America, 23: 1099, 1939.

44. Ricketts, H. T.: Problems connected with the use of protamine zinc insulin, Arch. Int. Med., 11:777, 1937.

45. McGee, L. A., and Martin, J. E., Jr.: The Hagedorn era in diabetes mellitus, West Virginia M. J., 35:361, 1939.

46. Smith, R., and Grisbam, W. H.: Survey of diabetes: statistical data and control comparisons with various insulins, Arch. Int. Med., 6:465, 1940.

47. Rolli, E. P., Brandaleon, H., and Fine, H. D.: Observations on the comparable effects of protamine zinc insulin and regular insulin in diabetes patients followed over a period of years, Ann. Int. Med., 16:750, 1942.

48. Reiner, L., Searle, D. S., and Long, E. H.: Insulin preparations with prolonged action—I. Globin insulin, Proc. Soc. Exper. Biol. & Med., 40:171, 1939.

49. Bauman, L.: Clinical experience with globin insulin, Am. J. M. Sc., 198:475, 1939.

50. Bauman, L.: Further experience with globin insulin, Am. J. M. Sc., 200:299, 1940.

51. Bauman, L.: The advantages and disadvantages of the old and newer insulins, Am. J. Digest. Dis., 12:23, 1945.

52. Andrews, G. B., Grant, W. A., Wood, A. V., and Jones, M. L.: Globin insulin, a clinical study, New York State J. Med., 913-917, 1940.

53. Duncan, G. G., and Barnes, C. E.: The action of globin insulin compared with that of crystalline, unmodified, and protamine zinc insulin, Am. J. M. Sc., 202:557, 1941.

54. Bailey, C. C., and Marble, A.: Histone zinc insulin, globin (zinc) insulin, and clear protamine zinc insulin: a comparative study of their action, J.A.M.A., 118:683, 1942.

55. Lawrence, R. D.: Globin-zinc-insulin: some experiments, Brit. M. J., 2:103, 1943.

56. Eaton, J. C.: Clinical trial of globin insulin and other insulins with delayed action, Lancet, 2:269, 1944.

57. Mosenthal, H. O.: Globin insulin with zinc in the treatment of diabetes mellitus, J.A.M.A., 125:483, 1944.

58. Martin, H. E., Simonsen, D. H., and Homann, N. H.: Time activity curves of globin insulin with clinical applications, Am. J. M. Sc., 208:321, 1944.

59. Trasoff, A., Barden, C., and Mintz, S. S.: Globin insulin: a clinical study, Am. J. Digest. Dis., 12:313, 1945.

60. Jackson, R. L., and McIntosh, C. B.: Globin insulin with zinc in the treatment of children with diabetes mellitus, Proc. Am. Diabetes A., 5:99, 1945.

61. Malins, J. M.: Globin insulin: clinical trial, Brit. M. J., 2:318, 1945.

62. Sindoni, A.: Present day conceptions of protamine zinc insulin and globin insulin with special references to insulin hypoglycemia and "insulin mixtures," Delaware State M. J., 18:21, 1946.

63. MacBryde, C. M., and Roberts, H. K.: Modified

protamine zinc insulin, an improvement on standard protamine zinc insulin, J.A.M.A., 122:1225, 1943.

64. MacBryde, C. M., and Roberts, H. K.: A modified protamine zinc insulin: comparison with histone zinc insulin, clear, and standard protamine zinc insulin, J. Clin. Investigation, 22:791, 1943.

65. Colwell, A. R., Izzo, J. L., and Stryker, W. A.: Intermediate action of mixtures of soluble insulin and protamine zinc insulin, Arch. Int. Med., 69:931, 1942.

66. Colwell, A. R.: Nature and time action of modifications of protamine zinc insulin, Arch. Int. Med., 74:331, 1945.

67. Colwell, A. R.: Effective insulin timing in diabetes mellitus, M. Clin. North America, pp. 327-341, March 1947.

68. Peck, F. B.: Approximate insulin content of extemporaneous mixtures of insulin and protamine zinc insulin, Ann. Int. Med., 18:177, 1943.

69. Peck, F. B.: Treatment of uncomplicated diabetes with mixtures of insulin and protamine zinc insulin, J. Indiana State M. A., 36:340, 1943.

70. Peck, F. B.: Treatment of diabetes by "single injection" method, West Virginia M. J., 40:341, 1944.

71. Peck, F. B.; and Schechter, J. S.: The newer insulin mixtures, a follow-up study, Proc. Am. Diabetes A., 4:59-80, 1944.

72. Peck, F. B.: Insulin mixtures and modifications, M. Clin. North America, pp. 343-357, March 1947.

73. Peck, F. B.; Personal Communication.

74. Ulrich, H.: Clinical experiments with mixtures of standard and protamine zinc insulin, Ann. Int. Med., 14:1166, 1941.

75. Wanchope, G. M.: Zinc protamine insulin and soluble insulin: interaction in combined doses, Lancet 1:962, 1940.

76. Sparks, M. T., and John, H. J.: Clinical use of mixtures of insulin, Ohio State M. J., 39:226, 1942.

77. Hildebrand, A. G., and Rynearson, E. H.: Clinical experience with mixtures of protamine zinc insulin and unmodified insulins, Arch. Int. Med., 72.37, 1943.

78. Lawrence, R. D., and Oakley, W.: Control of very severe diabetes by a new arrangement of insulins, Brit. M. J., 1:422, 1944.

79. del Fierra, R. S., and Sevringhaus, E. L.: Clinical use of new types of modified protamine zinc insulin, Ann. Int. Med., 22:667, 1945.

80. Aldersberg, D., and Dolger, H.: Insulin mixtures in the treatment of diabetes, J.A.M.A., 128:414, 1945.

81. Palmer, L. J.: Insulin in the treatment of diabetes mellitus, J.A.M.A., 132:502, 1946.

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THE INTERNATIONAL SOCIETY OF SURGERY (SOCIETE INTERNATIONALE DE CHIRURGIE) FOUNDED IN BRUSSELS, BELGIUM IN 1902.*

The announcement by the local committee of arrangements of the International Society of Surgery that this distinguished world-embracing association will hold its 13th international congress in New Orleans on October 9-15, marks an event of unusual interest and importance to the medical profession and people of this city.

*An introduction to the International Society of Surgery in happy commemoration of the 13th Congress to be held in New Orleans for the first time October 9, 1949.

DEFINITION, ORIGIN AND GENEALOGY

To avoid confusion with other surgical groups in America which claim to be of European parentage, a brief reference to the foundation and genealogy of this truly historical society is in order.

The International Society of Surgery was born in Brussels on September 9, 1902, under the auspices of the Belgian Society of Surgery with the collaboration of the leading surgeons of Europe and later of Britain, including Holland, France, Germany, Austria, Italy, and Spain, who had been invited to meet in Brussels to consider the creation of an international society of world-wide scope with its chief aim and purpose to organize world congresses, where the leading surgeons of the different countries could come together fraternally, in a common effort to contribute to the advancement of surgery, by the discussion and elucidation of their various surgical problems and scientific interests.

The Fellows of the Society were to meet in a spirit of scientific neutrality, free from nationalistic, partisan, racial, or religious prejudices. All the surgeons of Europe and America had realized by this time, at the bend of the century, that the time had come when surgery, profiting by the discoveries of Pasteur and of Lister, was making progress with gigantic strides, and that it was necessary to meet in international parliaments where the vast accumulations of new knowledge could be presented, appraised and stabilized for their definite value. The apparently assured political neutrality of Belgium, "guaranteed" by the great western European powers, in the famous treaty of London in 1839, (later proved to be a mere "scrap of paper," by the Germans when they invaded Belgium in 1914), seemed to make this a favorable country for a trial of difficult international organization. Fortunately the delegates of the different countries assembled in Brussels, agreed with their Belgian colleagues that the time had come for a trial of this enterprise and promptly approved the constitution and by-laws of the new society, with orders that the first international congress of surgery should be held in Brus-

sels, in the fall (September 18-22) of 1905. Coincidently with the adoption of the constitution, an international committee consisting of the delegates of the nations entering into the convention, was appointed with an executive committee at its head, consisting of The Belgian Quartet, Charles Willems of Ghent, Chairman, Antoine Depave, Secretary-General and Jules Lorthioir, Treasurer.

The Willems-Depage-Lorthioir Committee worked assiduously and with great earnestness, at their task; and so well, that by September 18, 1905, 26 different nations and 700 members were qualified and registered as members of the Society.

THE FIRST AMERICAN CONTINGENT

The United States was represented by 57 Fellows headed by a national committee consisting of Roswell Park (Buffalo, N. Y.), Chairman; J. Collins Warren (Boston); and Lewis McArthur (Chicago). The distinguished character and professional eminence of the American contingent was shown in the portrait gallery of the membership, published by Secretary Leopold Mayer, in the Transaction of 1926, where the American members were all, without exception, leaders and teachers of national reputation, each identified by some notable achievement in the domain of surgery.

This excellence of the American members, shared with those of other nations, was easily explained by the fact that admission to the Society could only be obtained by the approval of the national committee of each nation who passed on the merits of the candidates by their ethical and surgical reputation and achievements in their own countries. Besides the approval of the national committee the applicant would not be given full voting membership until he had been endorsed by the international committee at each congress.

The fact that the Society had been cast in an academic mold at its very birth, was shown by the limitation of its membership to a definite quota for each nation. The quota was revised at each congress and adjusted in proportion to its membership. The limitations of the quota and the strict observance of the standard of qualifications for admission, no doubt kept many worthy candidates from admission and led them to find relief for their ambitions by joining other societies in which no high fences were an impediment to easy entrance. The International Society has never aimed at *quantity* in its membership or at profit outside of its purely scientific and professional needs. It has always welcomed professional surgeons, who were capable of advancing the purposes of the Society by their learning and their capacity to contribute to the high scientific and professional merit of the organization.

THE ROYAL GREETINGS

The Society, from its very inception, was honored, encouraged and supported by the rulers and governments of the countries in which the congresses were held. The inaugural sessions in every one of these congresses have always been opened by the ruling head of the country whether a King, an Emperor, or a President. The eloquent speeches and the presence of the Ministers of State, Health, Education and Defense, in full uniforms, gave great brilliancy and impressiveness to these occasions.

THE ANCESTRY OF THE INTERNATIONAL SOCIETY

It is interesting to note that the fusion of the Belgian Surgical Society with the National Committee of the 26 nations that participated in the assembly of 1902, resulted in the formation of a new society which incorporated in its history all the ancestral surgical achievements and heroes of each one of the participating nations. In this way the International Society of Surgery may claim in the historical ancestries of its component nations, the names of the greatest masters of surgery in the world, as part of its own collective inheritance. Such are the lives of great and glorious searchers and discoverers as Andreas Vasalius (1514-1564), born in Brussels, father of human anatomy and the founder of rational surgery, based on an accurate knowledge of the human frame; Harvey, Hunter, and Lister in Britain, noblest of the immortal seers; Pare, Chauliac and Pasteur in France; Langenbeck, Graffe, Esmarch in Germany; Billroth in Austria; Boerhave and van Swieten in Holland;

Tagliacozzi, the father of plastic surgery, in Italy; Gimbernat in Spain; and last but not least, is the gift from America, the youngest in the line of its benefactions,—Morton and Long who discovered surgical anesthesia and, McDowell, the intrepid abdominal pioneer.

The genealogy of surgery proves that all the discoveries that constitute its greatness are largely borrowed from different countries and races.

PRESIDENTS

The presidents are elected by the International Committee of delegates from the different constituent nations. All the presidents have been historically identified bv some important general or special contribution to the advancement of surgery.

The Society had twelve congresses with the thirteenth under way and rapidly approaching. The first congress was held in Brussels (1905) under the illustrious presidency of Professor Theodore Kocher (1841-1917), of Berne—justly regarded as one of the safest and most skilful surgeons in the world. His clinic at Berne became the surgical mecca to which patients flocked from all parts of the globe, but particularly for the surgical cure of goiter. He performed over a thousand thyroidectomies with the lowest known mortality. His text book on operative surgery, translated into all languages was the current guide of most surgeons of his day. He was awarded the Nobel prize in 1909, for his researches in goiter. Dr. Halsted of Johns Hopkins was his close friend and admirer and spent many of his vacations at his clinic. Harvey Cushing worked in his laboratory and clinic in preparation for his future career as the leading neurosurgeon of his time.

Vincent Czerny (1842-1916) eminent surgeon, specially devoted to the study of cancer, at the head of his world renowned clinic at Heidelberg. Lucas Championiere (1843-1913) studied antiseptic surgery under the Master, Lister, in London, and introduced it in France. He was a pioneer in the treatment of fractures by early mobilization and massage to prevent muscular rigidity and atrophy; a pioneer in the surgical treatment of hernia and in many other surgical innovations. Antoine Depage (1862-1925), a powerful, tall man as well as intellectual giant, of great enterprise and originality in the building of hospitals and in devising new operative procedures especially applicable to military surgery and the author of the most complete bibliographic indices. President of the International Society in New York, 1914, he was hurried back to Belgium to direct the medical department of the Belgian Army, of which he was Surgeon General, which had been caught unprepared for an invasion without declaration of war. During the first World War he stood by the side of the heroic King Albert, of the Belgians, in holding a strip of shore on the edge of the North Sea, at LaPanne, where he built a hospital and refuge for the Belgian and allied wounded. There, Queen Elizabeth, the Royal Consort, aided by Mme. Depage, directed the nursing staff with a corps of patriotic Belgian scientists who improvised clinical laboratories for scientific work. Mme. Depage collaborated with the ill-fated Edith Cavell, the martyred English nurse, in establishing a home and training school for nurses. Mme. Depage appealed to American philanthropists for the relief of the allied wounded at La Panne. Meeting with a very generous response from America, she was returning home but met with a tragic end in the sinking of the Lusitania, by a German torpedo, off the coast of Ireland. Her body was identified and brought back to Belgium. Her husband, Dr. Depage, surgeon general of the Belgian Army, a broken-hearted man, soon followed; he died on June 10, 1925, and was buried beside her in the same grave. Both of these heroic figures are now in the same tomb, sharing the glory of their common patriotism.

After an interval of six years from 1914 to 1920, the first attempt to restore the activities of the International Society was made in Paris under the presidency of Professor W. W. Keen (1837-1932), Philadelphia,—first American president—pioneer in brain surgery and most prolific writer of classic surgical essays and editor of sur-

gical systems and text books. This meeting proved very encouraging and enthusiastic despite the deep resentment felt by the Fellows in the still lingering echoes of the furious anathema of the defeated German "intellectuals."

Eighteen years of prosperity followed, during which six international triennial congresses were held in succession; in London (1923) under Sir William MacEwen (1834-1924), the eminent surgeon of Glasgow and profound investigator of the endocranial infections; British science, the Crown and the Government were admirably represented. In Rome (1926), the 7th Congress was held under Professor Giordano (1864-), the universally known surgeon in chief of the City Hospital of Venice. This was a remarkable congress. It was notable not only by the beauty and dignity of its architectural surroundings and the excellence of the scientific program and very large and distinguished audience, but by (1), a marvellous tour of the Italian universities; (2), by a sonorous address, delivered in Latin, by the President—a great scholar as well as a master surgeon; (3), a most eloquent eulogy of the *surgeons*, by Sig. Benito Mussolini, the Premier and head of the Italian government, in which Il Duce confirmed his traditional oratorical fame; (4), an attempt to assassinate the Premier on leaving the Hall of the Congress. The attempt was thwarted by the quick action of Dr. Giordano, President of the Congress, who struck the arm of the would-be murderess (an insane woman), in time to deviate the course of the bullet from the temple to the nose, causing a grazing wound which did not prevent Mussolini from attending his official business the same day.

The following 8th Congress was held in Warsay (1929) under Professor H. Hartmann (1860-), of Paris; the 9th Congress in Madrid under Professor F. de Quarvain (1868-1940); the 10th Congress in Cairo (1935) under Dr. J. Shoemaker (1871-1940), of the Hague, who was called to the Presidency in replacement of Professor von Eiselberg, of Vienna, who was too ill to travel.

COMMENTARIES ON THE CONGRESSES

All these congresses were magnificent triumphs of the Administrative Bureau at Brussels, with Dr. Mayer, its guiding spirit, at its head. It is impossible to do justice to the wonderful collaboration and cordiality of our Colleagues in London, Rome, Warsaw, Madrid and Cairo, where the scientific programs were carried out, at times, under the strain from local political disturbances, as at Cairo; but always carried out most learnedly, conscientiously and profitably for all concerned. In all these congresses, the tourist excursions and sea cruises continued to be most valuable contributors to the pleasure and the educational profit of the congressists.

At Cairo, the International Committee realized that the time had come for the election of an American president. After several nominations, Dr. Rudolph Matas (1860-), Emeritus Professor of Surgery in the Tulane Universtiy of Louisiana, universally known for his original contributions to vascular surgery* was chosen and notified by cable, of his election *in absentia*. On receipt of his acceptance, his election was unanimously confirmed for the triennium, culminating with his presidency of the 11th Congress, of Vienna in 1938.

Unfortunately, the well organized plans for the congress in Vienna, were frustrated by the command of the Führer, Hitler, who forbade the Congress in German territory because the International Society admitted Jews to its membership. In consequence, and despite the protests and apologies of the Vienna surgeons and of the regrets of the German Surgical Society, the Vienna Congress was transferred to Brussels, its birthplace, where the Congress was received with a warm welcome by the profession and the government.

THE AMERICAN BRANCH OF THE INTERNATIONAL SOCIETY OF SURGERY

The dark clouds which had begun to gather over the 11th Congress at Brussels, con-

*Up to 1940, 640 operations on the main surgical arteries of which 260 were for aneurysms, with a gross operative mortality of 4.83 per cent. Annals of Surgery, vol. 112, Nov., 1940, pp. 802-859.

tinued to grow darker with the increasing Nazi persecution of the Jews, until the invasion of Poland in 1939 and the occupation of Belgium a few months later precipitated the explosion which shook the whole of Europe, including the frightful devastation in war-stricken Spain. So completely ruined were the countries involved in this second world war, that all the sources of professional knowledge and industry were completely suspended, including in this all the activities of the International Society. The Bureau at Brussels was excluded completely from all communication with the outside world during the German occupation of Belgium,—until the surrender of the German generals and deaths of Hitler and Mussolini, in 1945, put an end to the strife in Europe.

It was on November 1st, 1941, when the Nazi regime was at the height of its power over Belgium, that the Executive Committee of the American Branch of the International Society,—Drs. Elliott C. Cutler, Rudolph Matas and Eugene Pool, met at the Harvard Club in Boston on November 6, 1941, with other members, to consider plans of relief for the oppressed parent society in the grip of the enemy at Brussels. This was accomplished at a meeting held in the New York Academy of Medicine, November 12, 1942.

At this meeting a Council of Delegates representing all the branches of the Society in the Western Hemisphere, including North America, Mexico. Central and South America, and the West Indies. Dr. J. Arce, of Buenos Aires, Vice-President of the International Society since the Brussels Congress of 1938, was placed at the head of the Council and acted in this capacity until its dissolution at Cleveland on December 19, 1946. The Council appointed the United States National Committee (Cutler, Matas, Pool, Allen) who, acting under the authority of the Council continued to conduct the business of the Society until the automatic dissolution of the Council.

THE INTERNATIONAL SOCIETY IN AMERICA

As a result of the American administration with the authority of the Council of Delegates, the official continuity of the International Society was preserved until the liberation of Belgium permitted the Bureau at Brussels to resume its normal functions.

THE CORRELATION OF THE INTERAMERICAN CONGRESSES CONJOINTLY WITH THE CLINICAL CONGRESS OF THE AMERICAN COLLEGE OF SURGEONS

Unfortunately the greatest aim of the American Committee and of the council of delegates, which was to hold a polyglot, multilingual, surgical congress annually during the period of the war, conjointly with the yearly clinical congress of the American College of Surgeons, was completely nullified by the order of the War Department which advised against all meetings and great aggregations of people in large cities in line of communications. In this way the best opportunity to preserve the interest and loyalty of our members particularly those in the Latin American countries of the Western Hemisphere, was lost. On the other hand our North American Fellows who were members of the American Surgical and Southern Surgical Associations met regularly with these societies in their spring and winter sessions to discuss the reports of the Secretary-Treasurer, Dr. Matas, of the International Society of Surgery while keeping in touch with the scientific programs of the Society. While the United States National Committee met with the aforesaid societies, the most important work of correspondence and information was carried on in New Orleans at the office of Dr. Matas, Secretary-Treasurer.

Shortly after the organization of the American Committee for the relief of the Administrative Bureau at Brussels, the Committee was deprived of the very valuable collaboration of Dr. Cutler, who was transferred to London, as chief consulting surgeon of the armed forces of the United States at the Western Front; fortunately, he continued to serve as Chairman *in absentia*. Dr. Arthur W. Allen, also of Harvard, accepted the office and replaced Dr. Cutler up to the time of Cutler's greatly deplored death.

THE CHOICE OF NEW ORLEANS FOR THE THIRTEENTH CONGRESS

It is no doubt largely due to the remem-

brance of the long and arduous labors of the secretary-treasurer residing in New Orleans that members here and in Europe meeting in London decided to select New Orleans as the site of the next (13th) Congress. Though it is not unlikely that the International Committee in London, was influenced in the selection of New Orleans for the next Congress by sentimental reasons of gratitude for the secretary's faithful services during the whole period of the war, without expense to the Society, it would seem quite clear that the selection of New Orleans, the most cosmopolitan city in the United States, with its French and Spanish historic and romantic traditions had a strong appeal of its own, for its selection; besides, its constantly growing intercourse with the Latin American populations since the advent of aerial navigation and radio communications, is the most fit city, from the view point of the International Society's interest to receive and entertain the members of the 13th Congress, who were so largely representative of the Latin countries in Central Europe and the Mediterranean shores. Again, New Orleans could show by the side of the most precious relics of its French and Spanish architecture the latest and most approved hospital and other institutions for the sick and injured where the modern generations of surgeons, availing themselves of the latest equipments, are busy perpetuating and spreading the renown of their surgical ancestors.

NEW ORLEANS A MEDICAL CITY MADE PROSPEROUS AND HAPPY BY MEDICAL SCIENCE

By an unexpected and agreeable coincidence, the year 1905, which marks the date of the first International Congress in Brussels, the same year remains immortalized in the history of New Orleans as the year of the great victory of medical science over the yellow fever plague which, for a hundred years had kept New Orleans under its deadly domination. It was that year, when the surgeons of the world assembled in Brussels were celebrating the triumph of the Pasteurian doctrines and of the Listerian revolution, which had freed surgery from the tyranny of the septic microbes that people of New Orleans were celebrating the first decisive victory of sanitary science against "the yellow ogre of the Tropics," who for over a century had desolated Louisiana and the States bordering on the shores of the Gulf of Mexico, with the epidemic spread of its deadly visitations. Led by the teachings of Finlay of Havana and the heroic sacrifices of the United States Army Yellow Fever Commission (Reed, Carrol, Lazear, and Agramonte) the culprit virus-carrying mosquito, (*stegomyia fasciata*) was discovered and the methods and means for its extermination were applied by the illustrious Gorgas in 1900, at Havana. This success brought about the extinction of the *stegomyia fasciata* in New Orleans in 1905.

Forty-four years have elapsed since New Orleans, the worst plague stricken city of the continent, has been transformed into the healthy, prosperous, and ever-growing metropolis that it is today. Through this marvelous transformation it is possible to welcome the great company of the elect of surgery, who will soon arrive to receive our warm hearted hospitality, without fear of the infection of yellow fever, malaria, and other tropical pests, which would have made impossible the cordiality of our reception. Fortunately, "yellow jack" remains only the tragic memory of a long dead past.

The Congress held in London two years ago showed the wonderful recuperative power of the International Society in the face of adversity and in that exhibition of its robust vitality, it found its most stimulating encouragement in the example of indomitable courage, fortitude and equanimity of spirit amid the calamities of war, displayed by their hosts,—the British people and their surgeons,—that command the world's greatest respect and admiration.

SOME OF THE ACTIVITIES OF THE INTERNATIONAL SOCIETY

The Society publishes an official journal in which every article is summarized, at the end, in five languages of the congress.

The transactions of each congress appear usually in three bound octavo volumes, averaging from 850 to 900 pages each, with illustrations.

All the addresses, articles and discussions

are translated textually or in full abstract, in the official language of the congress. Besides the Transactions, a biographic and bibliographic Index Catalogue with more than 1,200 portraits of the Fellows of the Society was first published in 1933-1934, under the editorship of the Secretary General (L. Mayer), in two large octavo volumes. These were distributed in 1935 to all the members without extra charge. The Index-Catalogue is not only a valuable directory of "Who's Who" in the International Society as this is represented in the Fellowship of the Society, but it is also highly valuable as a classified bibliographic index to the 250,000 surgical pages that the Fellows of the Society have contributed to the professional literature up to 1935.* These contributions cover every phase of the vast technical and literary activity that has characterized the marvelous advance of surgery during the more than four decades that the International Society has been in existence, and no better commentary need be quoted on the scientific work of the Society than the fact that every outstanding and epochal advance in surgery has been contributed during this period by its members.

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*The publication of the Illustrated Biographic Index Catalogue of the Society and its members was interrupted by the second World War but preparations for the continuance of this invaluable publication have been resumed.

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Established 1844
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WHAT HAPPENED IN WASHINGTON

Important decisions were made in Congress in the past month. Two actions were taken in the interest of organized medicine and of the public, and these actions were taken in opposition to the desires of the most powerful political machine since the Civil War.

The first of these decisions was taken by the Ways and Means Committee to exclude physicians, dentists, and lawyers from the operation of a proposed expanded Social Security law. This action was taken after energetic presentations from various quarters were made to the Committee, and were largely assisted by our representative, Mr. Hale Boggs. A proposed law would levy a 2½ per cent tax on all self employed individuals on the first $4800 dollars of net income, or approximately $100 a year. However, the insured after the age of 65 could receive no part of this "insurance money," if he had any other income amounting to $14.99 a month, or more. In other words, 140,000 doctors would contribute $14,000,000 a year, of which the vast majority would receive no benefit whatsoever. The relief from such an unjust burden of selective taxation is a thing for which we thank the various spokesmen for organized medicine, the officers of the State Society, particularly Dr. Zander, and the Committee on Congressional matters, and Representative Boggs.

The second decision was even more important. It was the rejection by the Senate, with a vote of 60 to 32 of President Truman's reorganiaztion plan No. 1. Had the Senate not disapproved the plan it would have had the effect of submerging the governmental aspects of medicine to welfare projects of a cabinet post and elevating to that post Oscar Ewing, who is a vociferous advocate of compulsory sickness insurance—the apostle of State Medicine. The effects of this victory will be far reaching. In pleading for his cause before the Senate committee, Oscar Ewing stated that if the plan were rejected only noncontroversial plans can be put through. In other words, he recognized the issue as a test of strength, not only of the plan but of the whole project of building a political empire of welfare, medicine, insurance and security. The defeat of such a scheme is therefore equivalent to defeating compulsory sickness insurance and State Medicine for this Congress, and it may be expected as an issue in the Congressional election of 1950.

The medical profession should take cheer from this accomplishment, and each of us would do well to reflect upon how it was done and what it means. Up to the hour of voting in the Senate, the result was in doubt. In the weeks preceding August 16, various agencies within the realms of organized medicine had presented their reasons,

for opposing the President's plan, to members of the Senate and House. The officers of the State Society have been active in contacts with members of Congress. A meeting was held in Little Rock and another in Denver where plans were made for effective effort. Our committee on Congressional matters was represented in Washington by Drs. E. L. Zander and C. J. Brown. It has been stated that the physicians of the country, and the public, owe an unrealized debt to the citizen delegations from Arkansas, Oregon, California, Minnesota, Louisiana, Texas, Oklahoma, Kansas, and Virginia.

That the opponents of the plan took the issue seriously is shown by the words of Senator Humphrey (Democrat-Minnesota) who said that the A. M. A. was responsible for an adverse vote in the Committee and charged that if the plan "is killed because of the opposition of one lobby on behalf of a small group that is not even affected by it except in imagination, then we shall have offered proof that a democratic government cannot order its own affairs effectively. This is the test."

The conclusive majority by which the plan was killed was the result of education of the public on the topic of State Medicine and producing in the minds of the Senators the realization that the public knows what it wants. This is the culmination of years of activity and of ten months of intense effort. The way has been guided by the A. M. A. and state societies, but the work of local societies gave precision and point to the feelings of the doctors and the public.

With this in mind, never again should organized medicine take an attitude of disinterest where its affairs touch the public. We must ponder the vagaries of the leukocytes at the same time as we consider the endless contacts of our public relations. We should be aggressive in stating our position and in securing the adaptation of the problems of medicine to the needs of the public.

MEETING OF INTERNATIONAL SOCIETY OF SURGERY

New Orleans will be the focal point for a world-wide gathering of renowned surgeons, Octboer 10-15, 1949, when the International Society of Surgery will meet here.

This is one of the oldest surgical societies in the world, having been founded in 1902, and numbers among its members some of the foremost surgeons of the world, whose achievements are known not only to the medical profession, but whose names are known and revered by the lay public. One of these leaders who is thus famous is New Orleans' Dr. Rudolph Matas, who has been both secretary and president of the International Society of Surgery.

An excellent account of the history and accomplishments of this society, prepared in collaboration with Dr. Matas, will be found elsewhere in the Journal.

The Louisiana State Medical Society and its component organizations bids a welcome to the International Society of Surgery and hopes that the sojourn of its members in New Orleans will be not only instructive and profitable, but happy and enjoyable.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

REPORT OF CONFERENCE OF OFFICERS OF COMPONENT SOCIETIES

As representative of the Louisiana State Medical Society to the Fifth National Conference of Presidents and Officers of County Medical Societies on June 5 at Atlantic City, I wish to report a few highlights of the meeting.

At 9:00 a. m., a panel discussion concerning plans for handling emergency calls on

a 24 hour basis through different types of telephone answering services was held. This was followed by discussion of plans of various county and state societies for the care of indigent patients. Another panel discussion concerning responsibility of parish societies for community health programs and participation in the national education campaign was held. I cannot begin in a small space to give you the salient points of these discussions. However, I shall refer you to two booklets published by the Council on Medical Service of the AMA entitled, "Planning for Emergency Medical Calls", and "The Community Health Council". It would be worthwhile for every doctor sincerely interested in the welfare of his patients to study carefully and thoughtfully these two pamphlets. It is just as important today for all of us to be well versed in the socio-economic and political factors which affect our patients as with the strictly scientific phases of medical practice; for whether we appreciate it or not, he will be more influenced by our public relations technic than our scientific technic.

At the afternoon meeting the delegates were presented with a review of the first year of the British National Health Service in which was brought out the fantastic cost and inherent defects of the system.

Dr. George Lull explained the intimate relationship of the state medical societies to the AMA. In another paper, the importance of bringing allied organizations of dentists, nurses, receptionists, druggists and drug salesmen into closer association with our societies was stressed. Only in that way can the art of medicine, (harmonious relations) be developed to its maximum among our members.

Probably the most stirring presentation of the afternoon was a talk by the famous author Cecil Palmer of London, England, on the impact of socialized medicine on the British physician and his patient. Throughout his talk he stressed the fact that compulsory health insurance should be fought on a moral basis, strictly. For socialism in theory is very different from socialism in practice. To quote his words, "In Britain the doctors were winning all along the line, and it still is a mystery to me as it is a mystery to many of my fellow country-men and is a mystery to many members of the British medical profession—how it came about that at the eleventh hour, the medical profession gave up the ghost. I believe it was due to the fact that the Minister of Health in our present Socialist Government was able to divert the issue from the moral basis to the business. He was able to make the doctors, by a very clever political formula, discuss terms of service, whereas the doctors would have been on a stronger ground if they had said there were no terms of service under which they would degrade medicine by serving a state salaried medical service. I say the medical profession in Britain made a contribution to the servile state that had not been exceeded by any previous measure of nationalization in my Country. When the doctors were out and free, we had a chance; but when the doctors came in and made themselves the servants of the state—then we, indeed, had come to a position in which it would seem, at this moment, that we cannot possibly recover."

I. W. Gajan, Jr., M. D.

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OBSERVATIONS CONCERNING AMA MEETING

There is an experience which every doctor practicing medicine in America should have. It is as important a part of our medical education as the study of human anatomy. That experience is a first-hand study of the anatomy of your own House of Delegates at an AMA meeting. The sponsors of collectivism in America are trying to make you and me believe that our organization leaders are nearsighted men, interested only in their own aggrandizement. Spending some time at these business meetings will immediately dispel in our minds any idea of undemocratic procedure. All problems that intimately concern us as doctors and the American public who employ us, are openly discussed and passed upon by the doctors chosen directly by the rank and file. Before committee meetings there

were at the 1949 Session several laymen who presented views on problems facing us today. If your idea or plan of action is good you can be sure you will receive attention.

I do, like most of us, attend medical meetings to learn some new approach in the art and science of medicine. Much is gained and little is lost if each of us would spend a small portion of the time at business meetings of our representatives' bodies in order to better perfect ourselves in the art of medicine, so important today when our individual concepts of good scientific medicine are being so sorely pressed. No one understands, better than a physician, how important is continuous education in the best interest of those we serve (which includes us as human beings). When we learn that operation for a certain cancer is the best method of procedure we advise our patients and usually secure their permission. We must also learn the best therapeutic measures for the illnesses of the body politic which are intimately connected with psychosomatic medicine. For this knowledge we have no textbook or journal. It is only learned in the give-and-take and free exchange of ideas in our parish, district, state and national societies. If you and I do not inform ourselves and participate, we can blame no one but ourselves for foreign idealogies being thrust upon us. For whether we realize it or not, as a group we are an integral part of the most successful form of living the world has yet devised. Just as we never relax or "let Joe do it" in our constant fight on disease, we must never tire of supporting and encouraging the elementary principles of our form of living.

I. W. Gajan, Jr., M. D.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

DIABETES WEEK

OCTOBER 10-16, 1949

Dr. A. A. Herold, Chairman, has called a meeting of the Executive Committee of the Diabetes Association of Louisiana, for a date in September, to make arrangements for the Diabetes Week program. This committee consists of the members of the executive committee of the association who will be notified in advance of the right date.

The American Diabetes Association has scheduled an all year round diabetes detection program, which will be initiated with Diabetes Week, October 10-16, in the hope that a concentrated educational effort on the part of all will aid in discovering the one million undiagnosed and untreated diabetic patients in this country.

The American Diabetes Association has requested the cooperation of all state and local medical societies and asked that each medical society establish a Committee on Diabetes to make preparations for the drive.

The Diabetes Detection Drive was approved by the house of Delegates of the AMA on November 30, 1948 at St. Louis and again on June 7, 1949, at Atlantic City. It will require a concentrated effort on the part of all of us to discover these cases and then to secure treatment for the patients by their personal physicians.

SPECIAL NOTICE

There has been considerable misunderstanding as a result of the manner in wwhich the local press reported on the recent vote of the Senate when it defeated Truman's Reorganization Plan #1. The question before the Senate was the approval or disapproval of S.R. 147 which rejected Truman's Reorganization Plan #1. In this vote we should be particularly grateful to Senator Russell Long

for supporting the medical profession by casting his vote in favor of S.R. 147 and, in effect, against the Truman Plan.

SECOND DISTRICT MEDICAL SOCIETY

The Second District Medical Society met at the Metairie Country Club on August 18 with Dr. Jack Strange, President, presiding. Dr. Wm. H. Gillentine discussed use of digitalis in heart failure, based on a film of the same name supplied by Wyeth, Inc.

The next meeting will be held at the Metairie Club September 15 at which time colored films on common infectious childhood diseases will be presented.

ORLEANS CHAPTER
AMERICAN ACADEMY GENERAL PRACTICE OF LOUISIANA

At the June meeting of the Orleans Chapter of the A. A. G. P., Dr. Peter Graffagnino gave a most interesting and instructive discussion on "The Pitfalls in Obstetrics." His discussion was on:

1. The causes and treatment of rupture of the uterus in all types of pregnancy.
2. Review of bleeding cases and their control.
3. Toxemia of pregnancy.

We are indeed indebted to Dr. Graffagnino for giving us so much practical information in a few minutes and in a manner that was greatly appreciated.

At the July meeting the following officers were elected for the coming year:

President, Dr. Theodore F. Kirn; Vice-President, Dr. Albert B. Pitkin; Secretary, Dr. Vincent P. Blandino; Treasurer, Dr. Eugene Claverie, Jr.

Members of the Board—Dr. Nicholas J. Chetta, Dr. Alvin E. Johnson, Jr., Dr. Robert E. Gillespie, Jr., Dr. George D. Feldner, Dr. Charles V. Mosely, Dr. Cosmo J. Tardo.

The next regular meeting will be held in September and will be a dinner meeting.

INTERNATIONAL SOCIETY OF SURGERY

The scientific program of the thirteenth Congress of the International Society of Surgery, which will convene in New Orleans October 10-15, will consist of a symposium on the following subjects:

1. Surgery of the Parathyroid, Professor Raffaele Paolucci of Rome, Italy, will lead the discussion.

2. Surgery of the Pancreas. Professor Ian Aird of London, England, Professor of Surgery, University of London; Director of the Surgical Unit of the Post Graduate Medical School of London. (Successor to Professor George Grey Turner), will lead the discussion.

3. Treatment of Post Operative Thrombosis and Its Sequela. Professor Alton Ochsner, New Orleans, and Professor Michael DeBakey of Houston, will lead the discussion.

4. Surgery of the Pituitary Gland. Professor Paul Martin of Brussels, Belgium, Professor of Surgery in the University of Brussels will conduct this symposium.

5. Surgery of the Suprarenal Gland. Professor Rene Fontaine of Strasbourg, France, Professor of Surgery at the University of Strasbourg, will conduct this symposium.

6. The Causes of Recurrences after Operations on the Biliary Tract. Professor Rudolph Demel of the University of Vienna will conduct this symposium.

The last two days of the Congress will be devoted to American surgeons; this program will be prepared by Professor Evarts Graham, American Vice-President of the 13th Congress.

This program will consist of reports on Surgical development and research problems, along physiological and biochemical lines.

The list of those attending the Congress will include the most distinguished American, European, South American, and North African representatives. Among the distinguished guests will be Lord Alfred Webb-Johnson, President of the Royal College of Surgeons of England, Professor Rene Leriche, Clarence Craaford of Sweden and many others.

The Congress promises to be one of the high lights in New Orleans—already a city of distinguished gatherings.

The International Society of Surgery is forever linked with such great men as Theodore Kocher, Czerny, Lucas Championnere, Anton DePage, W. W. Keen, William Macewen, D. Giordano, Henri Hartment, Fr. DeQuervain, Shoemaker, Leopold Mayer, and our own distinguished Rudolph Matas, who presided at the Congress in Brussels, 1938.

The medical profession is invited to attend the scientific sessions of the Congress.

BIRTH REGISTRATION

In April 1945, the Louisiana State Registrar in cooperation with the Federal vital statistics agency, undertook an experiment in ten Louisiana parishes to determine if parent participation in the filing of birth certificates was desirable. The purpose of the plan was to place responsibility for filing birth certificates on mothers instead of the physicians. The test involved the use of a separate birth notification card to be completed and mailed to the local registrar by the physician or midwife performing the delivery.

The National Office of vital statistics very recently sent a representative on a visit to each of the ten parishes participating in the plan for the purpose of discussing the matter with the parish

health unit directors in order that it may properly evaluate the project.

The evidence at hand clearly indicates that the experiment did not produce results which would justify the introduction of this system on a statewide basis. In many instances physicians expressed resentment at the extra record work involved, and suggested that such a system was a reflection on their professional integrity.

Since no justification can be found for the additional work and expense that the continuation and furtherance of this project would involve, all Parish Health Unit Directors concerned were notified that the "Parent Participation Plan" of birth registration be discontinued immediately.

BOOK REVIEWS

British Surgical Practice: Edited by Sir Ernest Rock Carling, F. R. C. S., F. R. C. P., and J. Paterson Ross, M. S., F. R. C. S. (In 8 volumes.) Vol. 4. St. Louis, The C. V. Mosby Company, 1948. Pp. 486, illus. plates. Price per set, $125.00.

This book, which is volume 4 of a series of eight volumes on British surgical practice, is difficult to evaulate. In the first place, although the series constitutes, in effect, a system, the volumes are not loose-leaf and a system, whether or not it is called by that name, in which no provision is made for adding new material is likely to be out of date very soon. In the second place, the present volume, which covers alphabetical subjects from facial palsy to hiccup, includes a number of conditions of questionable surgical interest, such as filariasis and gout.

The most serious criticism to be advanced against this work, however, is the distribution of its available space. Fibrositis, for instance, occupies 10 pages, which is two pages more than the space allotted to gastrostomy and to hemorrhage and one page more than the space devoted to the general management of gunshot wounds. The latter presentation is exclusively military, but it is hard to see how it could be useful to any surgeon, whether he were in or out of the army.

Numerous similar examples could be cited, but the space allotted to hernia is perhaps the outstanding illustration of the disproportionate handling of the material. Twenty-two pages are given an excellent, profusely illustrated discussion of diaphragmatic hernia, which is certainly not a common surgical condition. All other varieties of hernia, inguinal, femoral, umbilical and incisional, as well as diastasis recti, are handled in 23 pages which contain only six illustrations (none strikingly good), all on inguinal hernia.

The list of editors of *British Surgical Practice* includes many eminent names. The material, within the spatial limitations, is generally well presented, though American surgeons, naturally, would not be in agreement with it upon all points. But the whole project seems of somewhat doubtful value. If volume 4 is typical, an experienced surgeon would not find the series at all useful and an experienced surgeon could not possibly gain enough from it to help him. The book is handsomely printed, but one cannot help wishing that some of the space wasted by the format—the very wide margins, to permit detailed marginal headings, and the style of the pages occupied by the index and listing the contributors and their titles—had been conserved and had been utilized to supply more information on the various subjects discussed.

FREDERICK FITZHERBERT BOYCE, M. D.

Current Therapy: Edited by Howard F. Conn, M. D. Philadelphia, W. B. Saunders Co., 1949. Pp. 672. Price, $10.00.

This book is a good addition to the armamentarium of the busy practitioner. To quote from the preface, "In this book for the first time an attempt has been made to furnish . . . not only the latest but a method endorsed and currently used by a competent authority."

Over 200 leading American physicians have contributed to this volume. The facts are presented briefly and pointedly. Several tables of references and a good index make fact finding a simple task.

The book measures 11" x 8" and the print is of a large, good type. This should greatly facilitate its use. It is a fine desk reference. It covers the

field of medicine and its allied specialties, and does it in a most satisfying manner.

I. L. ROBBINS, M. D.

Medicine Throughout Antiquity: By Benjamin Lee Gordon, M. D.; with a foreward by Dr. Max Neuburger. Philadelphia, F. A. Davis Co., 1948. Pp. 818, illus. plates. Price, $6.00.

Written for lay readers as well as for physicians, Dr. Gordon's book presents in an unusually interesting fashion the early development of medical thought from the days of prehistoric antiquity to the close of the Graeco-Roman period in 476 A. D. The medical practices of antiquity are shown as a link between the beliefs of ancient medical lore and the later centuries of increasing knowledge. Many fundamental discoveries of today are shown to be based on practices which antedate recorded knowledge. Translations of ancient manuscripts are included as evidence of this concept. The march of medicine is presented as a continuous forward movement, sometimes slow, sometimes in increasing tempo, but present progress ever based upon the knowledge of the past.

MARY LOUISE MARSHALL.

The Uses of Penicillin and Streptomycin: By Chester Scott Keefer. Lawrence, Kansas, University of Kansas Press, 1949. Pp. 72. Price, $2.00.

This book consists of three Porter lectures delivered under the auspices of the University of Kansas School of Medicine by an author well qualified by his broad experience in the field of antibiotics.

In the first lecture "Penicillin in Medical and Surgical Practice" attention is called to the remarkable importance which penicillin has assumed as a therapeutic agent within the few years since it has become available. The various types of penicillin, dosage forms and methods of administration are briefly reviewed. In discussing dosage the difficulty of making categorical statements in the light of present knowledge is recognized. It is pointed out however that organisms are most susceptible to penicillin when they are multiplying most actively. Thus, concentrations of penicillin within the body should be high enough to eliminate the most susceptible organisms within a short period of time and maintained sufficiently long for the more resistant organisms to recover and again to start multiplying rapidly. It is more important to maintain an optimal concentration for a prolonged period than a very high concentration for a short period of time although apparently optimal concentrations need not necessarily be continuous over a 24 hour period. This latter concept offers a rationale for the longer dosage intervals which have proved to be effective in some infections. In the author's opinion the widespread use of penicillin has not led to the production of a large number of penicillin resistant organisms and the risk of producing such strains seems to be minimal. The reactions to penicillin and the results of therapy in a variety of conditions are reviewed.

The second lecture is entitled "Streptomycin in the Treatment of Infections." The nature of the streptomycin complex and the chemistry of the important constituents are described. Information is provided on the susceptibility of various organisms and the occurrence of side reactions is discussed. The response to streptomycin therapy in various diseases is briefly considered with special attention being given to the therapy of tuberculosis.

The third lecture "Antibacterial Agents from Microbes" is of an historical nature concerning the work of Dubos, Fleming, Waksman and their co-workers in the development of tyrothricin, penicillin and streptomycin. It is of interest to learn that the demonstration of antibacterial agents from microbes dates back to the researches of Pasteur and Metchnikoff with other contributions of similar nature intervening between that time and the discovery of the agents used at present.

Although the limited treatment does not allow an exhaustive review of penicillin and streptomycin the reader will be well rewarded by an evening spent with this well written and informative book.

RALPH G. SMITH, M. D.

Doctors of Infamy: By Alexander Mitscherlich, M. D., and Fred Mielke. New York, Henry Schuman, 1949. Pp. 172, illus. Price, $3.00.

This is the story of the Nazi medical crimes with statements by three American authorities identi-

fied with the Nuremberg medical trial. The book is illustrated with 16 pages of photographs. The authors, who are Germans, voluntarily composed the book from authentic data and eye witnesses. The contents, in a general way, are known to most informed people, but it certainly behooves each and every individual to read this small book to realize the enormity of these inhuman crimes perpetrated in the name of medical science, and which American as well as all other scientists state were entirely devoid of any conclusions beneficial to the advance of medical knowledge.

I. L. ROBBINS, M. D.

Child Health Services and Pediatric Education: By American Academy of Pediatrics, Committee for the Study of Child Health Services. New York, Commonwealth Fund, 1949. Pp. 270. Price, $3.50.

The American Academy of Pediatrics has undertaken a program which has as its stated objective: "preventive, diagnostic, and curative medical services of high quality, which, when used in cooperation with other services for children, will make this country an ideal place for children to grow into responsible citizens." As the first step toward this objective a true picture of the present facilities for child health was necessary before intelligent positive action for improvement could be undertaken. This report is a factual summary of existing facilities.

The book is in two parts. Part I brings out the highlights of factual information related to private practice, hospital facilities and the services of community health agencies. Part II deals with an evaluation of pediatric education at the undergraduate, graduate, and post-graduate levels.

The average practitioner who is in any way concerned with any bureaucratic direction of his practice should be vitally interested in his attempt of a group of doctors to seek facts as to the adequacy of the present status of medical care for children.

JAMES U. MORRISON, JR., M. D.

Aesculapius Comes to the Colonies: By Maurice Bear Gordon, M. D. Ventnor, N. J., Ventnor Publishers, Inc., 1949 Plates illus. Pp. 560. Price, $10.00.

This story of the early days of medicine in the thirteen original colonies describes each medical condition as it existed prior to the American Revolution. Many pertinent documents dealing with local medical history are reproduced. Portraits and sketches of the principal physicians are included. An excellent index aids in making this volume a valuable reference tool for the further study of American medicine.

MARY LOUISE MARSHALL.

Neurological and Neurosurgical Nursing: By C. G. de Gutiérrez-Mahoney and Esta Carini. St. Louis, C. V. Mosby, Co., 1949. Pp. 516, illus. Price, $5.75.

The most remarkable thing about this book is that it actually was written for nurses. So often textbooks for nurses are nothing more than medical texts written down to what the author vaguely considers the nurse's "level." The author physician and his nurse co-author have presented only those facts concerning neurologic disorders which are essential for intelligent nursing. This book is not a text of neurology. The nurse is taught to practice nursing, not medicine. Instructions relative to spinal puncture, for example, are minute. With this book as a guide a nurse could set up for, and assist with, a spinal puncture without ever having seen one performed and without any concern about interpreting the results.

The nurse's role in the medical and surgical management of diseases of the nervous system makes up the body of the text. The appendix contains the outline for a comprehensive set of lectures including the history of neurologic nursing. A reading list, not the author's bibliography, as well as a glossary are appended. Any school of nursing could begin to teach neurologic nursing with a minimum of effort on the part of the instructor.

The book is cloth bound, of convenient size and amply illustrated. Nothing is left to the nurse's imagination—a sufficient recommendation in itself.

FREDERICK C. REHFELDT, M. D.

Psychosomatic Medicine: By Edward Weiss, M. D. and O. Spurgeon English, M. D. 2d ed., Philadelphia, W. B. Saunders Co., 1949. Pp. 803.

We can no longer deny the need for a good text on this all important problem. The first edition of this book was good enough to establish it as a

classic. This will be a "must" volume to those who would bring to bear "The clinical application of psychopathology to general medical problems."

I. L. ROBBINS, M. D.

PUBLICATIONS RECEIVED

The Blakiston Company, Philadelphia: Shearer's Manual of Human Dissection, Edited by Charles E. Tobin, Ph. D. Blakiston's New Gould Medical Dictionary, Edited by Harold Wellington Jones, M. D., Norman L. Hoerr, M. D., Arthur Osol, Ph. D.

The Commonwealth Fund, New York: Supplement to Child Health Services and Pediatric Education, Report of the Committee for the Study of Child Health Services, The American Academy of Pediatrics.

Grune & Stratton, Inc., New York: Progress in Neurology and Psychiatry, An Annual Review, Volume IV, Edited by E. A. Spiegel, M. D.

Harper & Brothers, New York: America's Health, by The National Health Assembly.

The Linacre Press, Inc., Washington, D. C.: Marihuana in Latin America, The Threat it Constitutes, by Pablo Osvaldo Wolff, M. D., Ph. D., M. A.

W. B. Saunders Company, Philadelphia: A textbook of Neuropathology, by Ben W. Lichtenstein, B. S., M. S., M. D.

Charles C. Thomas, Springfield, Ill.: Radiologic Exploration of the Bronchus, by S. DiRienzo, M. D.